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SURGERY

A Monthly Journal Devoted to the Art and Science of Surgery

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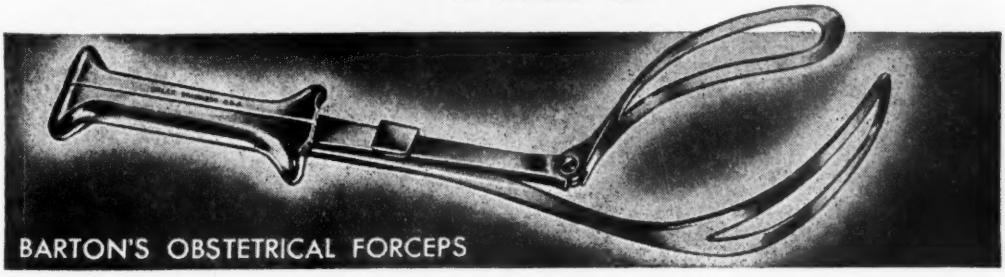

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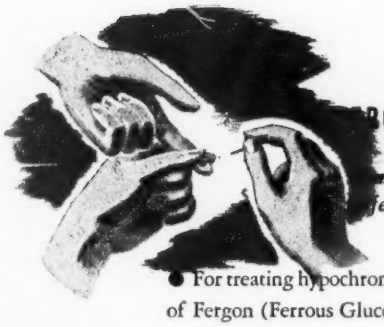

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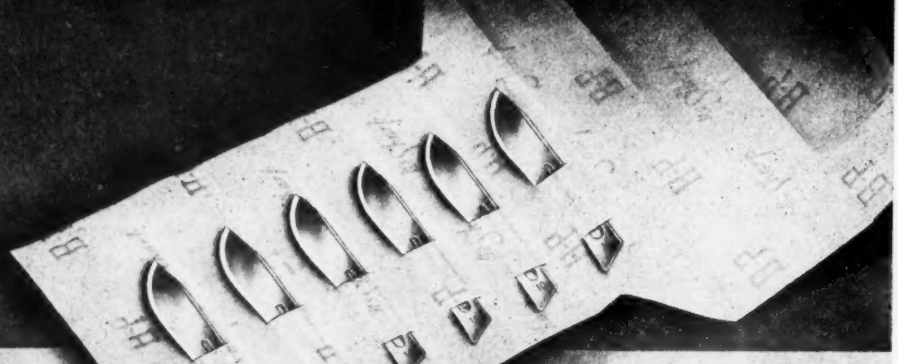

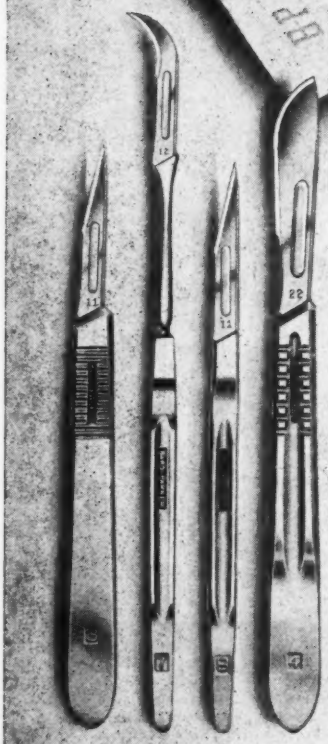

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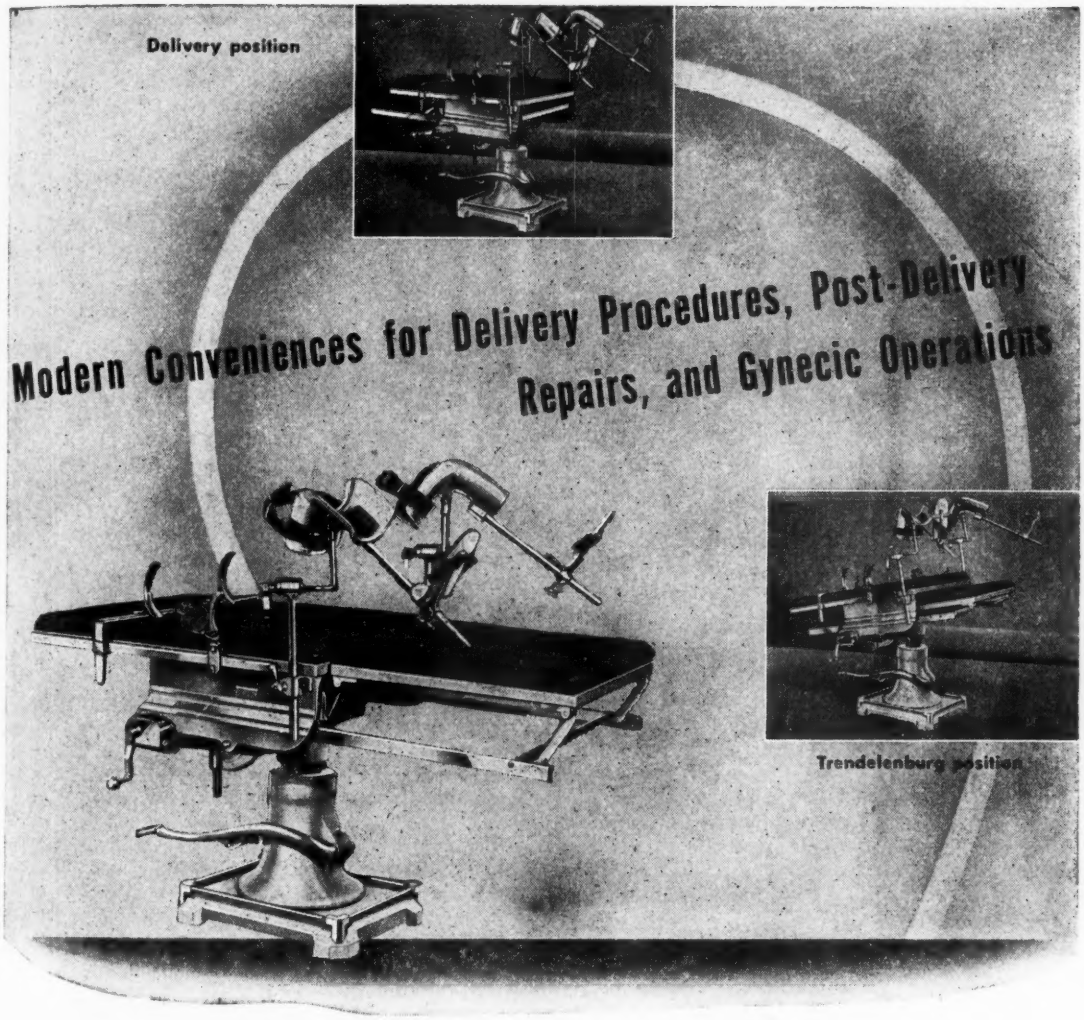
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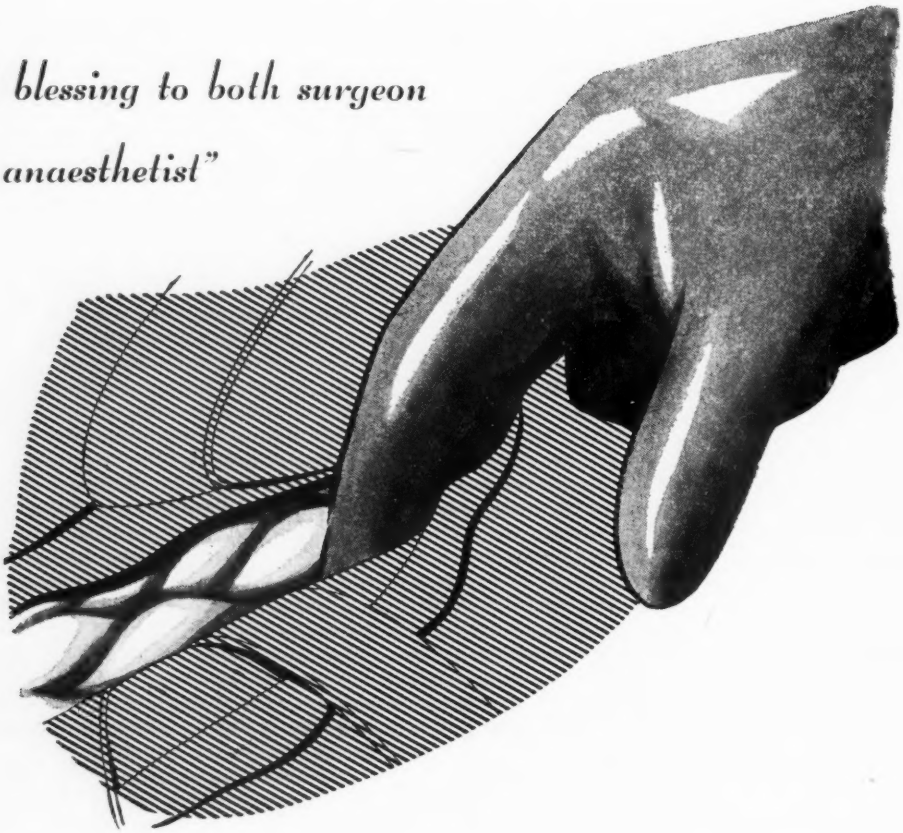

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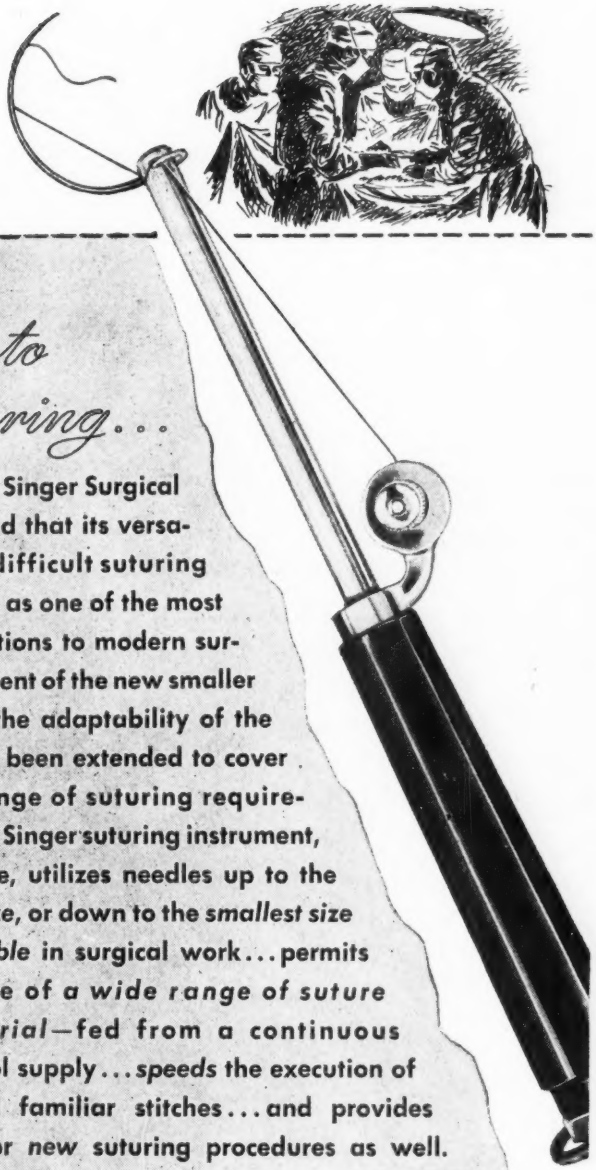

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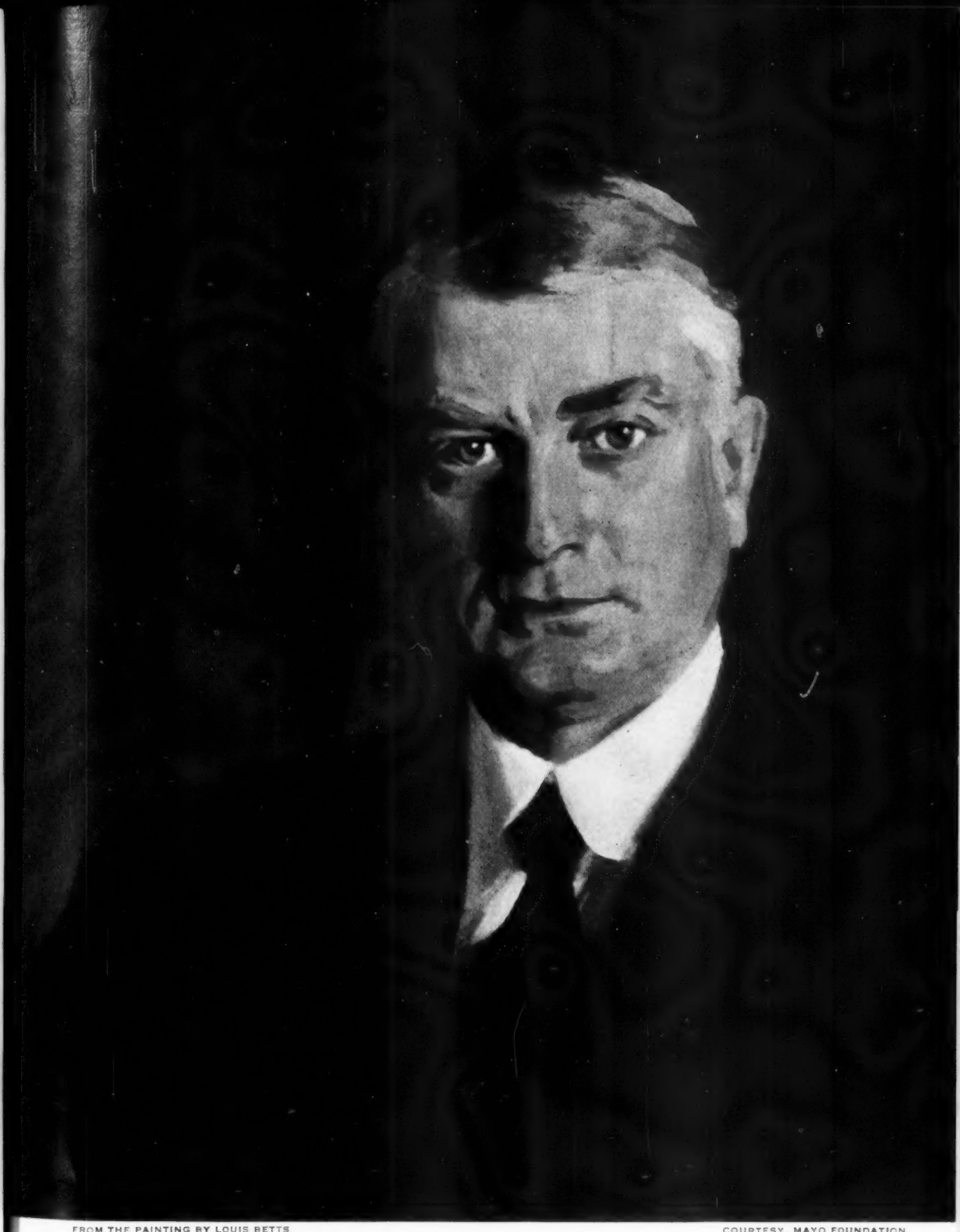

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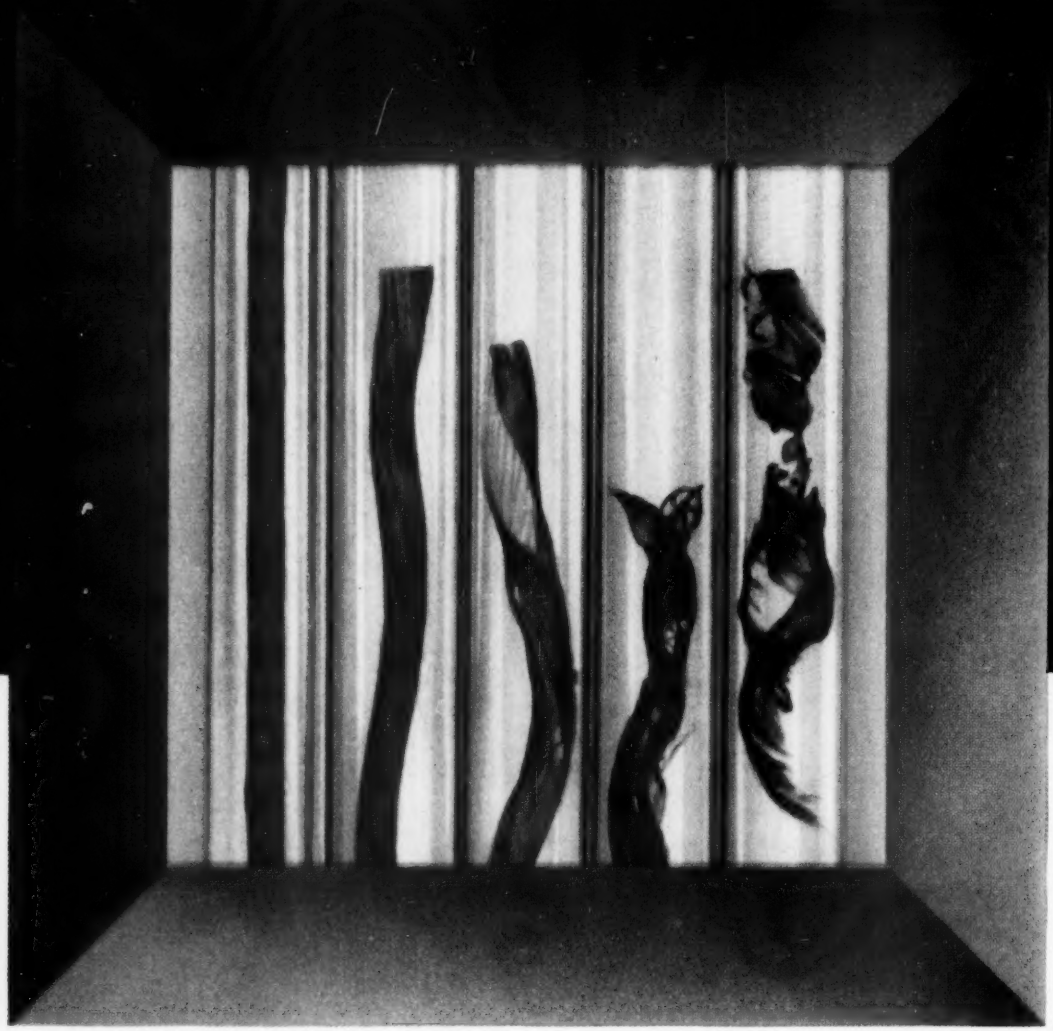
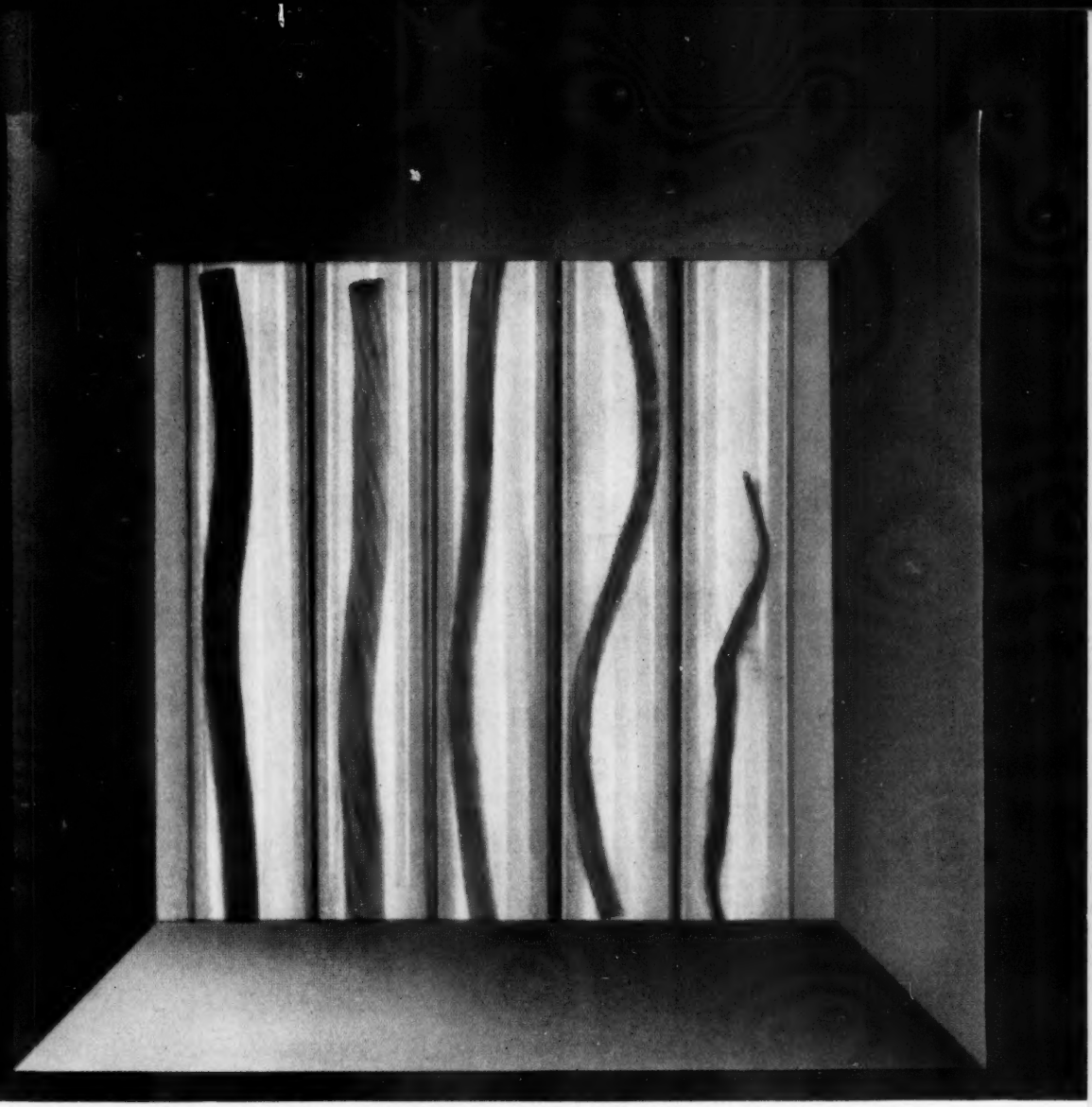


Enlarged photograph of five stages of digestion of surface-chromicized gut in trypsin solution.

SURFACE-CHROMICIZED CATGUT

THE MATERIAL: Surface-chromicized after spinning and drying. The chrome concentration is very high in the surface layers and relatively low in the core of the strand. THE RESULT: In enzyme solution, the core of most surface-chromicized catgut digests readily, leaving a hollow cylinder which separates into ribbons.

This cylinder may be excessively resistant to enzyme action and persist in tissue, frequently leading to knot extrusion.



Enlarged photograph of five stages of digestion of Tru-Chromicized gut in trypsin solution.

ETHICON TRU-CHROMICIZED CATGUT

THE MATERIAL: By the Tru-Chromicizing method, individual ribbons of catgut are soaked in chrome bath *before* they are spun into strand, permitting uniform deposition and full control of chrome concentration.

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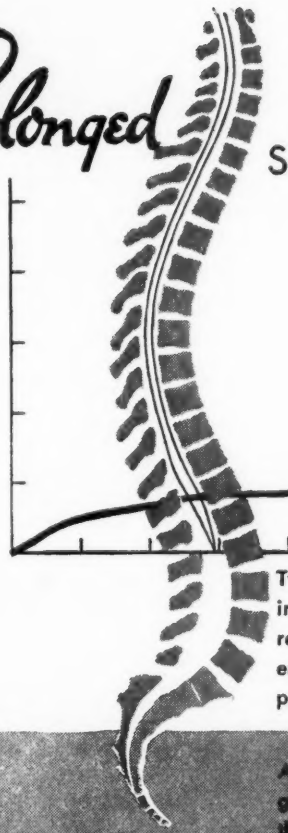

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[1]Coller, F. A. and Ransom, H. K.: Carcinoma of the Rectum. Conclusions Based on 12 Years' Experience with Combined Abdominoperineal Resection. Surg., Gynec. & Obst., 78:304,1944.

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SURGERY

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Original Communications

A REVIEW OF PATIENTS WITH INTRATHORACIC DISEASE AND INJURY TREATED ON THE SURGICAL SERVICE OF A UNITED STATES ARMY GENERAL HOSPITAL IN NORTH AFRICA

LIEUTENANT COLONEL JOHN R. PAINE, AND MAJOR ARTHUR G. PLANKERS, MEDICAL CORPS, UNITED STATES ARMY

INTRODUCTION

THIS report deals with 81 patients admitted to the Chest Division of the Surgical Service of a United States Army General Hospital during the period of its operation in North Africa during the spring, summer, and fall in 1943. This hospital's installations were all in Nissen huts and tents. Until the end of the Tunisian Campaign it was the most forward of any of the American general hospitals in North Africa. Due to its forward location and the recurring need for a large number of available beds to care for the casualties resulting from the bitter fighting of the last weeks of the war in Africa, it was impossible to keep some patients for periods longer than three weeks. The same thing was true, but to a lesser extent, at the time of the invasion of Sicily. Even at the busiest periods, however, the hospital was not called upon to give primary surgical treatment to any battle casualites except in a few isolated instances.

Only those patients with intrathoracic disease or injuries involving the thoracic cavity are considered in this report. Wounds involving the thoracic wall alone have been excluded since such wounds present no special problems not found in other wounds. Neither have patients with fractured ribs unassociated with intrathoracic complications been included. Many of the 81 patients had other wounds but these wounds did not appear to affect the course of the intrathoracic injuries, and for the purposes of this report have been neglected.

MISCELLANEOUS CASES

The 5 miscellaneous cases represented pathologic processes such as may be encountered in any general hospital in civil life.

One soldier presented a far-advanced fibrosarcoma of the chest wall which appeared to have followed an injury during training in the air-borne infantry. Biopsy of the tumor and aspiration of the chest to relieve dyspnea were the only procedures indicated.

Another patient was admitted with empyema of the right pleural cavity secondary to subphrenic abscess following a ruptured appendix. At the time

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TABLE I. CLASSIFICATION OF PATIENTS WITH INTRATHORACIC INJURIES OR PATHOLOGY

Cases of a miscellaneous character	5
Cases of nonpenetrating injuries	10
Cases of penetrating wounds	66
Total	81

of his admittance, the subphrenic abscess had been drained anteriorly. Rib resection and open drainage gave satisfactory immediate results.

Two patients with mediastinal tumors were seen. One of these tumors was thought to be a mass of partially calcified lymph nodes and was not operated upon. The second was operated upon and a teratoid tumor, approximately 10 cm. in diameter, was removed with considerable difficulty. During the closure of the thoracic wall a fatal hemorrhage occurred from the aorta at a point where the tumor had been closely adherent. The remaining 4 patients in this group were returned to the United States for further treatment.

CASES OF NONPENETRATING INJURY

Ten patients with nonpenetrating injuries of the thoracic cavity were encountered. Seven of these injuries were due to motor vehicle accidents and 3 were battle casualties. These patients were all treated initially at other hospitals and transferred to us during the early part of their convalescence. Other injuries present besides the chest injuries included lacerations, contusions, and cerebral concussion. Two patients with blast injuries from bomb explosions had large pneumothoraces. One of these pneumothoraces was of the tension type and necessitated continuous aspiration of the pleural cavity for eight hours at an evacuation hospital. Fractured ribs were present in all these 10 patients and in 4 instances ribs on both sides were involved. Hemothorax, pneumothorax, and subcutaneous emphysema were seen singly or in various combinations in all these cases.

Treatment at our hospital consisted only of strapping of the chest with adhesive tape when necessary to give comfort, and aspiration of the intrapleural blood and air when present in any but minimal amounts. One exception to this was a soldier who developed an empyema necessitating closed drainage.

None of the patients in this group died. Two were returned to duty sixty and sixty-two days, respectively, after injury. The remainder was evacuated to general hospitals further back or directly to the United States to complete their convalescence.

CASES OF PENETRATING INJURIES OF THE CHEST

Sixty-six patients were seen who had penetrating wounds of the thoracic cavity. These were all battle casualties, with three exceptions. A corporal (Case 11, Table V) was injured during battle training when a mortar shell fell short. A shell fragment penetrated through the right pleural cavity and lodged in the muscles beneath the right scapula. This patient was operated upon. Lacerations in the superior vena cava and right lung were sutured and a large quantity of blood removed from the right pleural cavity. A portion of this was returned to the patient by vein. A private (Case 12, Table IV) was stabbed by another soldier during the course of an argument. A lieutenant (Case 1, Table V) was accidentally stabbed during a volleyball game by the knife of a fellow player who had neglected to remove it from his belt.

TABLE II. PATIENTS IN WHOM INJURY WAS NOT CONFINED TO THE THORACIC CAVITY

INJURY	NO. OF PATIENTS
Lung and diaphragm	2
Liver and diaphragm	2
Lung, liver, and diaphragm	2
Total	6

This large group of patients from the standpoint of surgical therapy falls naturally into three classes as follows:

1. Patients treated without primary surgery other than débridement and without subsequent aspiration of the pleural cavity, 15.

2. Patients treated without primary surgery other than débridement and with subsequent aspiration of the pleural cavity, 33.

3. Patients treated with primary major surgery, 18.

In a somewhat rough manner this classification also divides the patients according to the severity of their injuries. There are, however, some exceptions. The large majority of patients with penetrating wounds of the thoracic cavity had had the wound of the chest wall débrided in the forward hospitals. In a few instances in which the wounds were small and the resulting injury minimal, no débridement at all had been done. No débridements were performed at our hospital in any of the chest cases except in the instance of case 11, Table V, mentioned previously. No wounds were of the sucking variety when seen at our hospital, although many were noted on the field medical record as being

TABLE III. PATIENTS WITH PENETRATING INJURIES OF THE THORAX TREATED WITHOUT PRIMARY SURGERY OTHER THAN SUPERFICIAL DÉBRIDEMENT AND WITHOUT SUBSEQUENT ASPIRATION OF THE PLEURAL CAVITY

CASE	CAUSE OF INJURY	TYPE OF INJURY	RETAINED FOREIGN BODY	RESULTS
1	Shell fragment	Laceration of lung, hemothorax, fractured rib, left	No	Evacuated, improved*
2	Mortar shell fragment	Laceration of lung	Yes	Returned to duty, recovered
3	Shell fragment	Laceration of lung	Yes	Returned to duty, recovered
4	Machine gun bullet	Laceration of lung	No	Evacuated, improved
5	Bomb fragments	Laceration of lung, hemothorax	Yes	Evacuated, improved
6	Mortar shell fragments	Laceration of lung, pneumothorax	Yes	Evacuated, improved
7	Shell fragment	Laceration of lung	Yes	Evacuated, improved
8	Shell fragment	Laceration of lung, hemothorax	Yes	Returned to duty, recovered
9	Mortar shell fragments	Laceration of lung	Yes	Evacuated, improved
10	Rifle bullet	Laceration of lung, pneumothorax, fractured ribs, right	No	Evacuated, improved
11	Mortar shell fragment	Laceration of lung	Yes	Returned to duty, recovered
12	Rifle bullet	Laceration of lung, pneumothorax	No	Evacuated, improved
13	Shell fragments	Laceration of lung	Yes	Returned to duty, recovered
14	Shell fragment	Laceration of lung, hemothorax	No	Evacuated, improved
15	Shell fragment	Laceration of lung, hemothorax	No	Evacuated, improved

*This patient developed a localized empyema at the site of exit of the fragment which also shattered the rib. A resulting bronchial fistula was closed and the empyema cavity obliterated by operation.

TABLE IV. PATIENTS WITH PENETRATING INJURIES OF THE THORAX TREATED WITHOUT PRIMARY SURGERY OTHER THAN SUPERFICIAL DÉBRIDEMENT AND WITH SUBSEQUENT ASPIRATION OF THE PLEURAL CAVITY

CASE	CAUSE OF INJURY	TYPE OF INJURY	RETAINED FOREIGN BODY	COMPLICATIONS	AMOUNT OF FLUID ASPIRATED (C.C.)	RESULTS
1	Shell fragments	Laceration of lung, hemothorax, fractured rib, right	No	None	345	Returned to duty, recovered
2	Shell fragments	Laceration of lung, hemothorax	Yes	None	3,520	Evacuated, improved
3	Shell fragment	Laceration of lung, hemothorax	No	Empyema, bronchial fistula	650	Evacuated, improved
4	Shell fragment	Laceration of lung, hemothorax	No	None	1,150	Evacuated, improved
5	Shell fragments	Laceration of lung, hemothorax	No	Empyema, bronchial fistula	1,400	Evacuated, improved
6	Machine gun bullet	Laceration of lung, hemothorax, fractured rib, left	No	None	More than 100	Returned to duty, recovered
7	Rifle bullet	Laceration of lung, hemothorax, pneumothorax	No	None	1,110	Returned to duty, recovered
8	Shell fragment	Laceration of lung, hemothorax, pneumothorax, fractured ribs, left	No	None	1,400	Evacuated, improved
9	Rifle bullet	Laceration of lung, hemothorax, fractured ribs, left	No	None	1,000	Evacuated, improved
10	Machine gun bullet	Laceration of lung, hemothorax, pneumothorax	No	None	700	Evacuated, improved
11	Shell fragment	Laceration of lung, hemothorax	Yes	Coagulation of intrapleural blood with beginning organization	2,675	Evacuated, improved
12	Knife	Wound of liver, diaphragm, and lung, hemothorax	No	None	1,700	Evacuated, improved
13	Machine gun bullet	Laceration of lung, hemothorax, pneumothorax, fractured rib, left	No	None	2,155	Returned to duty, recovered
14	Machine gun bullet	Laceration of lung, hemothorax, pneumothorax	No	None	915	Evacuated, improved
15	Rifle bullet	Laceration of lung, hemothorax, pneumothorax	No	None	800	Evacuated, improved
16	Pistol bullet	Laceration of lung, hemothorax, pneumothorax, subcutaneous emphysema, fractured clavicle, right	No	None	2,970	Returned to duty, improved
17	Pistol bullet	Laceration of lung, hemothorax, pneumothorax	No	None	1,320	Returned to duty, recovered
18	Shell fragment	Laceration of lung, hemothorax, pneumothorax, fractured rib, right	No	Coagulation of intrapleural blood with beginning organization	2,060	Returned to duty, recovered
19	Shell fragment	Laceration of lung, hemothorax	No	None	350	Evacuated, improved
20	Shell fragment	Laceration of lung, hemothorax	Yes	None	50	Evacuated, improved

TABLE IV—CONT'D

CASE	CAUSE OF INJURY	TYPE OF INJURY	RETAINED FOREIGN BODY	COMPLICATIONS	AMOUNT OF FLUID ASPIRATED (C.C.)	RESULTS
21	Machine gun bullet	Laceration of lung, hemothorax, fractured rib, left	No	None	650	Evacuated, improved
22	Shell fragment	Laceration of lung, hemothorax	No	None		Evacuated, improved
23	Rifle bullet	Laceration of lung, hemothorax	No	None		Evacuated, improved
24	Shell fragment	Laceration of lung, hemothorax	Yes	None	2,400	Evacuated, improved
25	Bomb fragment	Laceration of lung, hemothorax	No	None	1,775	Returned to duty, recovered
26	Machine gun bullet	Laceration of lung, hemothorax	No	Empyema, bronchial fistula		Evacuated, improved
27	Shell fragment	Laceration of lung, hemothorax, pneumothorax	Yes	None	700	Evacuated, improved
28	Shell fragment	Laceration of lung, hemothorax	Yes	Coagulation of intrapleural blood with beginning organization	2,000	Evacuated, improved
29	Shell fragment	Laceration of lung, hemothorax, fractured rib, left	Yes	None	700	Evacuated, improved
30	Machine gun bullet	Laceration of liver and diaphragm, hemothorax	No	Empyema		Evacuated, improved
31	Rifle bullet	Laceration of lung, hemothorax	No	None		Return to duty, recovered
32	Shell fragment	Laceration of lung, hemothorax	Yes	Empyema, bronchial fistula		Evacuated, improved
33	Shell fragment	Laceration of lung, hemothorax, pneumothorax, severance of spinal cord	No	Empyema, bronchial fistula	3,140	Evacuated, improved

such initially. Ribs were frequently noted to have been fractured by the penetrating missles but this complication provided no special therapeutic problem in any of our cases.

As can be seen from Tables II to V, bomb, mortar, and shell fragments were by far the most frequent causative agents with machine gun, rifle, and pistol bullets accounting for the remainder of the battle casualties.

Thoraco-Abdominal Injuries.—According to a strict interpretation of the term, 6 patients might be classified separately as having thoraco-abdominal wounds, but this has not been done. In only 4 of these patients was the liver injured and in every instance the injury was slight. There were no other intraperitoneal injuries. The injury to the diaphragm in all 6 cases was not great. None of these patients died or developed complications secondary to the wounds of the diaphragm or liver while under our observation.

1. Patients Treated Without Primary Surgery Other Than Débridement and Without Subsequent Aspiration of the Pleural Cavity

There were 15 patients in the group treated without primary surgery other than débridement and without subsequent aspiration of the pleural cavity. In some of these cases hemothorax, pneumothorax, or both were present in a minimal degree. Convalescence proceeded satisfactorily and aspiration was not

TABLE V. PATIENTS WITH PENETRATING WOUNDS OF THE THORAX TREATED WITH PRIMARY MAJOR SURGERY

CASE	CAUSE OF WOUND	TYPE OF WOUND	OPERATION	RETAINED FOREIGN BODY	PLEURAL SPACE DRAINAGE	FLUID ASPIRATED FROM PLEURAL SPACE AFTER OPERATION (C.C.)	RESULTS
1	Knife	Laceration of diaphragm and intercostal artery	*Thoracotomy:* Suture of diaphragm and ligation of intercostal artery	No	No	250	Returned to duty, recovered
2	Bomb fragments	Laceration of lung, fractured rib, left	*Thoracotomy:* Excision of foreign bodies and suture of lung	Yes	No	Not known	Evacuated, improved
3	Shell fragment	Laceration of lung, fractured rib, right	*Thoracotomy:* Suture of lung	Yes	No	Not known	Evacuated, improved
4	Shell fragment	Contusion of lung, fractured ribs, left	*Thoracotomy:* Exploration	No	Yes	75	Evacuated, improved
5	Shell fragment	Laceration of lung	*Thoracotomy:* Suture of lung	Yes	No	1,200	Returned to duty, recovered
6	Shell fragment	Laceration of lung, fractured rib, right	*Thoracotomy:* Partial lobectomy	No	No	25	Evacuated, improved
7	Rifle bullet	Laceration of lung, subcutaneous emphysema	*Thoracotomy:* Excision of foreign body and suture of lung	No	No	900	Evacuated, improved
8	Shell fragment	Laceration of lung	*Thoracotomy:* Suture of lung	Yes	No	485	Returned to duty, recovered
9	Mortar shell fragment	Laceration of lung	*Thoracotomy:* Excision of foreign body and suture of lung	No	No	Not known	Evacuated, improved
10	Shell fragment	Laceration of lung	*Thoracotomy:* Excision of foreign body and suture of lung	No	No	1,770	A.W.O.L., recovered
11	Mortar shell fragment	Laceration of superior vena cava and lung, fractured sternum	*Thoracotomy:* Suture of superior vena cava and lung	Yes	No	250	Evacuated, improved
12	Shell fragment	Laceration of lung	*Thoracotomy:* Excision of foreign body and suture of lung	No	Yes	Not known	Evacuated, improved
13	Shell fragment	Laceration of lung, subcutaneous emphysema	*Thoracotomy:* Excision of foreign body and suture of lung	No	Yes	Not known	Evacuated, improved
14	Shell fragment	Laceration of lung, liver, and diaphragm	*Thoracotomy:* Excision of foreign body and suture of diaphragm and lung	No	Yes	Not known	Evacuated, improved
15	Mine fragments	Laceration of lung and diaphragm	*Thoracotomy:* Suture of diaphragm and lung	No	No	Not known	Returned to duty, recovered
16	Mine fragments	Laceration of lung	*Thoracotomy:* Suture of lung	Yes	No	440	Evacuated, improved
17	Bomb fragments	Laceration of lung	*Thoracotomy:* Suture of lung	Yes	Yes	Not known	Evacuated, improved
18	Shell fragments	Laceration of liver and diaphragm	*Laparotomy:* Suture of diaphragm	No	No	Not known	Evacuated, improved

performed. Despite the presence of retained metallic foreign bodies in the lungs of 9 of these patients, no operative procedure for their removal was done. Complications developed in only 1 instance. This soldier developed a localized empyema with a bronchial fistula which had to be closed by operation. Five patients of this group were returned to duty sixteen to seventy-one days after receiving their wounds. The remainder was evacuated to the rear areas or to the United States. No patient died.

2. Patients Treated Without Primary Surgery Other Than Débridement and With Subsequent Aspiration of the Pleural Cavity

Thirty-three patients received penetrating wounds of the thorax not of sufficient magnitude to necessitate any primary surgery other than the débridement of the wound of the chest wall performed at the forward hospitals, but developed pneumothoraces and hemothoraces which required aspiration. In some cases only one aspiration was performed, while in others several aspirations were carried out. The total quantity of bloody fluid removed from the chests of these individuals varied from 50 to 3,520 c.c. The air, often removed simultaneously with the fluid, was not measured. In only 8 cases were there retained foreign bodies and no operations were performed at our hospital for their removal. Six of the 33 patients developed empyema and in 5 of these 6 there was an associated bronchial fistula. In only 1 case of empyema was there a retained foreign body.

In 3 cases an intrapleural hematoma became organized. Each of these patients was subjected to thoracotomy, the organizing hematoma removed, and the lung freed.

None of the 33 patients died. Nine patients were returned to duty after periods of treatment up to ninety-five days. The remainder was evacuated to other base hospitals or to the United States for further treatment.

3. Patients Treated With Primary Major Surgery

The 18 cases in which primary surgery was performed for the intrathoracic wound included 17 instances of thoracotomy and 1 instance of laparotomy. Only one of these operations was performed at our hospital. This patient (Case 11, Table V) has been mentioned previously. The usual procedure at the time of operation was to ligate all bleeding vessels and suture lacerated lung tissue. Only 1 patient had had a partial lobectomy. When received at our hospital, 7 of these patients had retained intrathoracic metallic foreign bodies demonstrable by x-ray examination. Thirteen patients were operated upon and cared for without postoperative drainage of the thoracic cavity. The other 5 were drained by catheter for two to four days after operation. Two patients developed complications: one (Case 9, Table V) had a severe wound infection in the chest wall, and another (Case 11, Table V) developed an intercostal neuritis which necessitated cutting two intercostal nerves proximal to the thoracotomy incision.

No patient died. Four were returned to duty at intervals up to forty-one days after injury. One patient left the hospital without leave fifty-two days after his injury. The remainder was evacuated to the rear or to the United States for prolonged convalescence.

DISCUSSION

Since almost all the patients with thoracic injuries were admitted to our hospital by way of transfer from more forwardly situated evacuation and

station hospitals, some two to fourteen days after being wounded, we were rarely called upon to determine how such patients were to be treated. Definitive treatment had already been started. By and large the patients arrived in good general condition considering the seriousness of their wounds. This speaks well for the large volume of work performed under trying circumstances by the evacuation hospitals. In most cases all that we could do was to ensure, as far as possible, the continuance of a satisfactory convalescence and to return to duty as many men as possible, physically fit to withstand sustained hard physical exertion.

To fulfill these duties properly, considerable thought was given to three problems. These were:

1. The treatment to be employed for accumulations of blood in the pleural cavity
2. The treatment to be employed for retained intrathoracic foreign bodies
3. Criteria to be employed in determining whether or not a patient was fit to return to duty

Six months is indeed a short time in which to answer such questions from our own somewhat meager experience but some decision which would serve as a working basis had to be found immediately.

We had been greatly impressed in 1942 by the results obtained in England by Mr. Tudor Edwards and his colleagues in the treatment of hemothorax following wounds of the chest by repeated aspiration. These men advocated repeated frequent aspirations, begun thirty-six to forty-eight hours after the occurrence of the hemorrhage and continued until no further fluid accumulated in the pleural space. Such a regimen seems justified by the facts, as demonstrated by Tudor Edwards, that early aspiration did not produce additional bleeding and that the incidence of infection was much lower in those patients in whom the pleural space was kept free of fluid by aspiration than in those treated conservatively, that is, without aspiration. An additional reason for the persistent use of aspiration is the very real danger of coagulation and subsequent organization of an intrapleural collection of blood.

A thoracentesis was performed in each case of thoracic wound or injury in which intrapleural fluid other than a minimal amount was thought to be present either by physical or x-ray examination. Routine use was made of the efficient portable electric suction pumps provided by the Army Medical Corps. The degree of suction produced by these machines can be quickly and accurately regulated by a control screw, and six months' experience has shown that the machines are safe when used carefully. Most of the patients were aspirated every third or fourth day as long as was required by the reaccumulation of the fluid. No arbitrary limit was set as to the quantity of fluid or air, or both, that should be removed at one time. The patient's subjective complaints of tightness, pain in the chest, or the onset of coughing were taken as indications to terminate the aspiration. On no occasion, however, were more than 900 c.c. of fluid removed at one time. The usual aspirations averaged between 400 and 600 c.c.

As practically all of our patients had been injured more than forty-eight hours before reaching the hospital, there seemed to be little danger of initiating fresh bleeding by the disturbance of intrathoracic pressure relationships due to thoracentesis. In only a few instances, therefore, was air injected following the aspiration and then only for the increased comfort of the patient. No instances in which aspiration produced renewed bleeding were noted.

From the information available, we feel unable to express any opinion as to the effect of aspiration in the development of the 9 cases of empyema present in this series. No case of empyema was seen to develop at this hospital, however, as a sequel to thoracentesis. All of those patients noted as having empyema had the infection when first admitted to the hospital. The incidence of empyema following wounds that penetrated the pleural cavities was approximately 11 per cent (7 out of a total of 66 wounds).

To illustrate the typical and satisfactory response to thoracentesis in a suitable case, the story of one soldier (Case 24, Table IV) can be told briefly.

CASE REPORT

This soldier experienced a penetrating wound of the left side of the chest from a small shell fragment Sept. 12, 1943, near Salerno, Italy. The skin wound was small in size, being 1 cm. in length and irregular in contour. The shell fragment was retained within the thoracic cavity. The wound in the chest wall was sucking in character. This was closed with one mattress suture as an emergency procedure at a field dressing station the day of the wound. In addition, the patient was given 1,000 c.c. of plasma. Following this he lay in a barn at the Salerno beach head for two days before being evacuated directly to our hospital by hospital ship and ambulance.

At the time of his admission to the hospital, Sept. 20, 1943, the left lung was totally collapsed and the mediastinum shifted to the right. Fluid and superimposed air filled the left pleural cavity. The patient was dyspneic and pale with a temperature that ranged between 101 and 103° F. Thoracenteses were performed September 21, 22, 24, 26, and 27 with a total of 2,400 c.c. of bloody fluid and a large quantity of air being removed. This fluid was found to be sterile by culture on three occasions and while containing approximately 35 per cent hemoglobin at the first aspiration, became more serous in character at each aspiration. The temperature remained elevated for four days until all sulfonamide drugs were omitted. These had been given in large doses irregularly since the day he had received the wound. Subsequent therapeutic trial proved him sensitive to these drugs.

This patient made a rapid recovery. By Sept. 27, 1943, he was up and about without complaints. X-ray examination of the chest was negative except for the presence of an opaque foreign body 3 by 10 mm. in size in the hilar region of the left lung. Oct. 15, 1943, he was evacuated to the rear to complete his convalescence.

No patient was operated upon for the removal of foreign bodies retained within the parenchyma of the lung as determined by anterior-posterior and lateral roentgenograms, although 26 of the 67 patients with penetrating wounds had such a complication when they were admitted to the hospital. These foreign bodies were all small, being less than 10 mm. in their greatest diameter as estimated from the x-ray film, except in two or three instances. In these exceptional cases the largest diameter varied from 10 to 15 mm. Which foreign bodies should be removed, and when, we are not qualified to say. None of the 26 patients with this complication that were observed by us presented symptoms which could be definitely attributed to the foreign body, however.

Since the exigencies of war allowed some patients to remain in the hospital only a few days while others remained a relatively long time, no accurate impression of the proportion of soldiers with intrathoracic injuries, such as those contained in this report, that can be returned to duty in a given time is possible. Information, of course, is also lacking on how well those soldiers that were returned to duty held up to the strain of army service. Nevertheless, it is interesting to note that approximately one-fourth (20 out of 76) of the patients with serious thoracic injuries were returned to service. For only 7 of these 20 patients was a limited type of duty requested. These 7 all had penetrating wounds.

There was a total of 66 patients with penetrating wounds of the chest. Forty-eight patients with penetrating wounds were therefore evacuated to the

rear or returned to the United States as class "C" casualties. The patients placed in class "C" at our hospital numbered 11. There were 37 patients evacuated to hospitals further in the rear in North Africa. We feel certain that a number of those evacuated to the rear could have been returned to duty after a few additional weeks of convalescence and probably were so handled. We have no information, however, as to what actually did happen to any of the evacuated patients. Of the 11 patients placed in class "C" at our hospital, 3 were so classified solely because of wounds other than of the chest. In the 8 remaining cases the chest wound was the only, or one of several reasons, for placing the patient in class "C."

It may be questioned whether we were justified in returning men to duty as quickly as we did. During the early weeks that the hospital was in operation no definite criteria were followed. The decision rested entirely on the discretion and clinical judgment of the surgeon. During the latter weeks, however, after a spirometer had been constructed out of tin cans, string, and solder, vital capacities were recorded on a few patients and it was tentatively decided that despite the patient's general condition and despite the fact that all his wounds had healed, no soldier would be returned to duty unless he had a vital capacity of at least 3,500 c.c. Whether such a rule will be of help or not, future experience will have to say. Any means by which the decision to return a wounded man to the dangers and rigors of battle can be made more objective should be welcomed by all military surgeons.

Special mention should be made of the 3 patients in whom coagulation and beginning fibrosis of the intrapleural accumulations of blood occurred. Such a result occurs much more frequently than has generally been believed in the past. Thoracotomy with removal of the clot and decortication of the lung has become the accepted procedure in such cases. Two of our patients with this condition were operated upon elsewhere. The history of the third patient is reported here.

CASE REPORT

A soldier (Case 28, Table V) was struck by shell fragments, Sept. 13, 1943, near Paestum, Italy, with resulting penetrating wounds of the right arm, right knee, and left side of the chest. Fourteen hours later, at an evacuation hospital, his wounds were debrided and the abdomen explored through a laparotomy incision. The abdominal cavity was found to be normal. Two hundred cubic centimeters of blood were aspirated from the left side of the chest. One week later he was transferred to a British general hospital where frequent attempts to aspirate the chest were made. On only two occasions were sizable amounts of fluid recovered.

This patient was admitted to our hospital Oct. 4, 1943, at which time all of his wounds were found to be healed. The left side of the chest was dull to percussion, silent to auscultation, and moved very little during respiration. X-ray examination revealed opacity throughout the entire left lung field with a marked shift of the mediastinum to the right. A small opaque foreign body could be detected in the hilar region of the left lung. Several attempts at aspiration at different places in the chest yielded only small amounts of differently tinted fluid.

Oct. 11, 1943, the left seventh rib was resected and the pleural cavity opened widely. The left lung was found to be entirely collapsed and the left pleural cavity filled with an organizing hematoma which contained scattered locules of fluid. This material was all removed and a layer of organized fibrin, 5 to 10 mm. thick, was then stripped from off the visceral pleura. This allowed the lung to expand and fill the chest completely when the incision was closed. The pleural cavity was not drained, but aspirated at six, twenty-four, and forty-eight hours after operation with recovery of 75, 30, and 5 c.c. of bloody fluid, respectively. The patient made a very satisfactory convalescence, being out of bed and walking about at the end of one week. Oct. 26, 1943, he was returned to the United States for further convalescence.

SUMMARY

Eighty-one patients with intrathoracic disease and injury were treated on the Surgical Service of a United States Army General Hospital during its service in North Africa. Sixty-six of these patients were battle casualties. One patient died during operation for a mediastinal teratoma. Patients with intrapleural accumulations of blood or bloody fluid were treated by repeated thoracentesis. Seventeen patients had been subjected to primary major surgical procedures at more forward hospitals. Only one patient was so treated at our hospital. Twenty-six patients had metallic foreign bodies retained within the chest. None was operated upon at the hospital. Twenty-five per cent of all patients with intrathoracic wounds or injuries were returned to duty during their stay at the hospital. One patient with an organizing intrapleural hematoma was successfully operated upon with gratifying immediate results.

PUTRID EMPYEMA WITHOUT FOUL SPUTUM

"Surprise" Putrid Empyema

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PUTRID empyema usually is a complication of a putrid pulmonary abscess. Most putrid empyemas are readily recognized as such before pus is obtained and examined because they occur in patients with known pre-existent abscesses with foul sputum. In another paper* we have pointed to the frequent occurrence of empyemas complicating "shut-off" putrid lung abscesses. In those cases an abscess of substantial size is the usual precursor of the empyema. There remain instances of putrid empyema in which sputum when present is not foul and a causative pulmonary abscess is not demonstrable. We have termed these cases "surprise" putrid empyemas because they are often not recognized, and frequently not suspected, before a pleural aspiration is performed. Excluded from consideration in this paper are foul empyemas derived from subphrenic, mediastinal, or other nonpulmonary sources.

Putrid empyema without foul sputum is not to be classified as rare merely because the cases do not appear as such in the literature. They have been encountered not infrequently in our experience but only fifteen well-documented cases have been selected to form the basis of this paper.

The age and the sex incidence are of interest and each has a bearing on the subject. There were 2 women and no children. The youngest patient was 28 years of age while most were in the fifth decade.

Careful history and meticulous examination are necessary in order to discover the etiology of the pleural infection. Two cases occurred after general anesthesia for unrelated conditions. One followed tooth extraction, one after removal of tartar from the teeth, and another after tooth repair under local anesthesia. In one case there was a three-day interval between the removal of

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*Stats, D., and Neuhof, H.: Putrid Lung Abscess Without Foul Sputum; Shut-off Lung Abscess, Arch. Int. Med. (In press.)

nasal polyps and the onset of the disease. In the other cases there were no obvious etiologic factors. In 5 of these the teeth and gums were healthy; in 6 specific mention was made that gingivodental infection existed. There were no tonsillar infections or infections elsewhere in the body; sepsis was not present. Coma or dulling of the senses other than normal sleep had not been present. The great similarity of all these facts to the situation in "shut-off" acute putrid abscesses and to a lesser degree in typical acute putrid abscesses will be pointed out later.

The clinical manifestations have been broken down into several headings:

1. Onset and Course: In 10 of the 15 cases the onset was sudden with moderately or extremely severe chest pain. This was characteristically aggravated by movement, respiration, and often by pressure on the chest and was typical, severe, pleuritic pain. In some the pain remained severe for several days and gradually tapered off but never completely disappeared. In others the pain diminished in severity rather quickly only to reappear with increasing severity. In 4 cases the pain lasted for about one week and then disappeared. In most instances, however, pain persisted until the time of operation (several weeks). In the remaining 5 cases pain was delayed for varying periods after the onset. In 1 case (see Case Reports) of chronic putrid empyema, pain first appeared about one and one-half months after the onset but persisted in mild fashion until operation about two months later.

In 10 of the 15 cases, cough was not present when the disease began. In 4 cases there was no cough at any time during the entire course. In 9 cases cough was mild or moderate and in 2 was severe, starting several days to a week or more after the pain first appeared, persisting for a varying time often until the postoperative period and then diminishing. In 3 cases cough was present from the onset. In only 2 instances was the cough severe. Both of these patients had bronchiectasis probably unrelated to the empyema.

Absence of significant amounts of expectoration parallels that of cough. In 4 there was no sputum; in 3 there was scant expectoration for a few days; in 4 scant expectoration lasted for one week or longer, and in the remainder moderate amounts were brought up for longer periods. Streaks of blood were unusual; hemoptyses did not occur; foulness was never present. The sputum was either mucoid and saliva-like or mucopurulent.

Nine of the 15 patients were moderately or severely and critically ill at, or shortly after, the onset. In one the onset was mild but the symptoms became severe after one week. The other 4 cases presented pictures of relatively mild illness at the onset and throughout the course. In these the patients were ambulatory for varying periods after the inception of disease and the temperature ranged between 100 and 102° F.

2. Physical Examination: The presence of fluid in the pleural cavity was suspected in most cases on admission. The general appearance of critical illness was often present. Dyspnea or tachypnea and cyanosis were present in about three-quarters of the cases. The condition of the teeth and gums was mentioned under etiology. When the pleural fluid was situated between the parietal pleura and the lung, dullness to flatness and diminished to absent fremitus and breath sounds were the usual findings. With the fluid in an unusual location as subsequently ascertained (infrapulmonary, interlobar, or paramediastinal), the findings were confusing and difficult to interpret. Tenderness of the chest wall was extreme in several cases. Clubbing of the digits was encountered only three

times in this series. In 4 instances the pleural fluid and air accumulated so rapidly and in such large volume that displacement of the trachea and mediastinal structures occurred.

3. Physical examination of the chest has been relegated to a subsidiary position as roentgen aid to diagnosis has developed. The roentgenographic appearance in these cases of putrid empyema was that of localized or more widespread collections of fluid with or without the presence of air. In some the initial appearance suggested a simple effusion. This was due usually to the outpouring of sterile sympathetic fluid as was shown in a number of the cases by aspiration of the chest. Later in the course, with the persistence or sudden reappearance or exacerbation of the chest pain, the picture was often quite different. Thus, a putrid pyopneumothorax was frequently disclosed representing a rupture of the pulmonary abscess into the pleural cavity. The roentgenographic picture may change suddenly. Any new incident such as cessation of cough, reappearance of pain, sudden rise in temperature, or increase in prostration should be checked with a film. A simple effusion may be transformed into a pyopneumothorax. The x-ray appearance may differ but little from, or be identical with, that of "shut-off" putrid pulmonary abscess with empyema described in another paper. An underlying pulmonary abscess was never demonstrable roentgenographically in this series. The difficulties mentioned elsewhere in interpreting correctly the shadows due to loculated infrapulmonary, interlobar, or paramediastinal pyopneumothoraces pertain also to these cases.

4. In order to convey a picture of the difficulty of diagnosis, reference should be made to the number of chest aspirations that were resorted to in these cases before foul pus was obtained. In only 3 cases did the first chest aspiration in the hospital reveal foul pus and in all there was present an unusually widespread putrid pleural infection. In the other cases an average of four chest aspirations each was required before foul pus was obtained and the diagnosis established. One or more of three reasons can be invoked to explain the repetition of aspiration which was necessary before pus was obtained: (1) Inaccurate roentgenographic localization of the pleural collection, (2) sympathetic effusions which often preceded and masked the underlying putrid infection, (3) unusual situation of the empyema which is characteristic of many of these cases. We can add here the fact that the collection of foul pus may not only be situated at a point away from the surface but also may be small. Hence, whenever a putrid empyema is suspected (not in obvious cases) aspiration should be performed in the operating room and the needle left in place for the operative procedure. There have been not a few instances in which great difficulty has been experienced in the operating room in finding foul pus which previously had been revealed by aspiration.

5. Closely related to the number of chest taps is the delay which occurred from the time of hospital admission both in ward and private patients until the true nature of the pleural infection was ascertained. Excluding those cases in which a chest tap on admission revealed foul pus, there was a delay of seventeen days, on the average, from admission to diagnosis and operation. When to this is added two- to three-week periods or longer (one case three months) during which time patients remained at home or in another hospital, evidently it has taken long periods in most cases for the diagnosis to be established. We will indicate later that most of these patients were considered to have some other intrathoracic disease throughout most of the clinical course.

6. The treatment of putrid pleural infections is always surgical. The details of the operative procedure of drainage of these putrid empyemas will not be given here. Some basic considerations are:

A. Before rib resection is undertaken, an aspirating needle should be in situ in the empyema. This will enable the operator to locate the putrid infection. The exception to this is a situation in which foul pus has been found previously, but cannot again be reached by an aspirating needle on the operating table. Cautious search must then be made after a portion of a rib has been excised.

B. In critically ill or moribund patients in whom large empyemas exist, local anesthesia is imperative and in rare instances only preliminary intercostal tube drainage is permissible. This may so improve the patient's condition that a more extensive procedure can be undertaken later.

C. In other patients, one or more ribs are excised to provide adequate access. The contents of the empyema cavity are evacuated by suction and sponge. The full limits of the cavity are ascertained. Occasionally, counter-drainage of the cavity will be necessary. The entire space is packed lightly with iodoform gauze. It is important to incise and excise the tract of the aspirating needle that penetrated the empyema in order to obviate a phlegmon of the chest wall. The visceral pleura is inspected for a lung abscess.

7. Following these principles the operation disclosed empyema cavities of various sizes, filled and covered by a shaggy necrotic foul exudate. In a few cases a lesion could be discerned in the pulmonary parenchyma but in none was a lung abscess found at operation. A blowing bronchial fistula was demonstrated at operation in only 1 case. In 1 case the empyema cavity contained about 100 c.c. of pus; in 6 cases between 200 and 300 c.c. was found; in 6 instances up to 1 liter was present and in the remaining 2 the lung was completely collapsed and approximately 2 liters of fluid were removed. The empyema cavities were usually of bizarre shapes. Extensions from the main cavity to form narrow tracts or tongues were not infrequent. Special technical problems often arise because of the relatively frequent occurrence of interlobar, paramediastinal, or infrapulmonary collections.

8. The postoperative course usually was smooth and convalescence was rapid when adequate drainage and exposure of all the recesses of the empyema were carried out. There were 3 deaths in the series of 15 selected cases due essentially to delay in diagnosis. One occurred in a patient who had been at home for three weeks after the onset, treated as a case of myocardial infarction. Autopsy performed two weeks after drainage of the putrid empyema failed to disclose any evidence of a residual putrid infection. The heart and coronary arteries were relatively normal. Death was due to bronchopneumonia. Another patient was treated at home for two weeks for lobar pneumonia. On admission to the hospital, she had a total putrid pyopneumothorax which was drained. The cause of death was a putrid mediastinitis and pericarditis. The third case was a 70-year-old patient who was treated under a mistaken diagnosis of myocardial infarction, pneumonia, and pleural effusion. Eighteen days after the onset, the fluid was withdrawn in order to relieve respiratory embarrassment. The operator was greatly surprised to obtain foul pus. Despite adequate operative drainage at this late date, the patient eventually succumbed to a nonputrid bronchopneumonia. At autopsy there was no evidence of a putrid empyema, lung abscess, or myocardial infarction.

Most of the patients showed improvement within several days after drainage. Depending upon the previous duration of disease and the ease of exposure of recesses, the temperature was normal after one to two weeks. The empyema cavities became clean in a few days. In 3 of the 15 cases a transient bronchial fistula was noted in the first or second week after operation.

The differential diagnosis of these "surprise" or "silent" putrid empyemas was often difficult. The diagnosis should always be entertained whenever an adult presents the features which have been described. When the suspicion is aroused, the aspirating needle will provide the evidence that a putrid infection exists. Aspiration of foul fluid or air is the only means by which the diagnosis can be made in these cases. On the other hand, aspiration has its dangers and must be employed carefully and with roentgenologic control. A list of the diagnoses which were entertained before the correct diagnosis was made will indicate the difficulties which were present: lobar pneumonia, bronchopneumonia, bronchiogenic carcinoma, pleural effusion, nonputrid empyema, peripheral type of pulmonary carcinoma, actinomycosis of the lung, and tuberculosis.

What is the evidence that putrid pleural infections which fit into the syndrome described are due primarily to a ruptured acute putrid pulmonary abscess?

By reference to the clinical data of "shut-off" putrid lung abscess in another paper, one will be struck by the great similarity between them and the ones detailed here. When a "shut-off" putrid lung abscess implicates the pleura and is complicated by a putrid empyema, its clinical picture may be identical with that of "surprise" putrid empyema described in this paper. There is the same prominence of severe prolonged chest pain and the same deemphasis on cough and expectoration. The cases may be similar clinically; they respond to the same therapy. Then, there are the cases of putrid empyema, in which an abscess cannot be demonstrated unequivocally, in which a shallow depression in the visceral pleura may be observed at operation. These depressions may represent the sites of ruptured lung abscesses. Reference should be made to those cases in which a lung abscess has not been demonstrated but in which a bronchial fistula was noted during the postoperative course. Such a fistula, even when open for a short time, must indicate dissolution of pulmonary tissue and the related bronchus, presumably a lung abscess. The cases in which there is good reason to believe that some pulmonary lesion did exist differ in no way from the other cases of putrid empyema in which such evidence is lacking. The entire group bears a strong clinical resemblance to "shut-off" acute putrid pulmonary abscess with empyema.

We have become convinced, after careful observation at operation of these and other cases, that the size of ruptured "shut-off" putrid abscesses with empyema is not the same as before rupture. It is very likely that putrid contamination of the pleura occurs only by actual and fairly free rupture of the pulmonary lesion. At times we have been able to see abscesses with their roofs blown off, so to speak. At other times a bead or large droplet of pus on the visceral pleura may indicate the presence of an underlying abscess. When the rupture occurs, it appears that in certain instances the abscess tends to collapse. When such an abscess is opened surgically, numerous fairly large bronchi may be found in the wall. This can be interpreted as showing that the volume of pulmonary tissue originally involved was large. Considerations and observations of this type lead us to expect a diminution in the size of a putrid lung abscess when it has spread to the pleura; that is the abscess collapses after rupturing.

Roentgenographic studies afford other evidence of the existence of bronchopleural communications in some of the cases of "surprise" putrid empyema. Such communications can be interpreted as showing the previous presence of a lung abscess. On several occasions, we have observed the accumulation in the pleural cavity of large quantities of air in a matter of a few hours. The successive roentgen observations in such cases reveal the transformation of small pleural effusions into pyopneumothoraces. Even if the bacteria in putrid infections were gas producers, it is inconceivable that 1 liter or more of gas could be generated within a period of several hours. Since we have never observed gas in a putrid abscess in a location in which entrance of air from the outside could be excluded, we believe that the bacteria involved in such an infection are not gas producers within the human body. We interpret the fluid levels observed in hydro- or pyopneumothoraces as evidence of bronchopleural communication and indirectly of a pre-existent lung abscess.

Finally it must of course be assumed, in the absence of other sources, that the pleural infection was derived from the lung, either a pulmonary abscess or a gangrenous bronchopneumonia. If an underlying pulmonary suppurative focus is not postulated as the cause, one must assume seepage into the pleura from a pulmonary lesion of whose existence there is no acquaintance.

CASE REPORTS

W. W. (Hospital No. 431467), was a 46-year-old man whose admission was antedated by a three-month history of malaise, low-grade fever, sweating, and a mild cough. Two months before admission he had a small hemoptysis and this represented about all the expectoration. About one and one-half months before admission he experienced sudden, severe, sharp pain in the right side of the chest, which subsided somewhat in the next few days but persisted mildly until admission.

He was well nourished. Marked pyorrhea alveolaris and clubbing of the fingers were present. Physical examination of the chest revealed a circumscribed area of flatness over the right upper posterior part of the chest and roentgenographically this was a homogenous, fairly dense, well-delimited shadow whose nature was not clear. At bronchoscopy, a slight amount of thin, nonfoul, nonrecurring secretion was noted from the right upper lobe bronchus. Aspiration of the pleural cavity was attempted on five occasions and was unsuccessful. Two weeks after admission, after a course of three and one-half months of low-grade chronic illness, a chest aspiration revealed typical foul fluid.

Immediate operation through the last needle tract disclosed an exceedingly thick-walled flat putrid empyema. A definite communication with pulmonary parenchyma could not be demonstrated. The empyema was opened widely and all recesses were packed lightly.

The cavity diminished in size rapidly and the patient became asymptomatic. Two months after operation the wound was completely healed and the patient was asymptomatic. At follow-up examination two years later the patient was well.

This is an unusual case of chronic putrid empyema without evident underlying putrid lung abscess. The prolonged chest pain, mild cough, and absence of sputum are typical.

L. J. (Hospital No. 393858), a 29-year-old man, had a tooth filled under local anesthesia, two weeks before admission. One week before admission he experienced sudden, severe, stabbing pain in the left lateral part of the chest. This continued until admission. There was no cough or expectoration. Low-grade fever and moderate weakness were present.

Examination revealed an acutely ill patient. There was dullness and diminished breath sounds over the left lower lobe. Clubbing of the digitis was not observed. At no time was there any expectoration and slight cough was present for only one day. The temperature varied between 102 and 104° F. and the chest pain diminished gradually. X-ray examination on several occasions revealed a small amount of fluid at the left base. Fourteen days after the onset the beginning of clubbing of the digits was observed. Two aspirations of the chest failed to disclose foul fluid but the third, twenty days after the onset, revealed foul pus.

One liter of thin putrid pus was evacuated at operation. It was situated paravertebrally. A primary pulmonary source was not seen. The postoperative course was smooth; the cavity

became clean in about one week and all symptoms disappeared. A bronchial fistula did not appear. Three months after discharge the wound was healed and the patient was asymptomatic at follow-up examination four years later.

This is the typical sequence in a case of putrid empyema in which a pulmonary source was never demonstrated. It is assumed that a small acute putrid lung abscess ruptured early in the course and was responsible for the empyema.

J. E. (Hospital No. 486600), a 70-year-old man, became ill three days before admission with sudden, severe, pain in the left anterior part of the chest, cough, temperature to 101° F., and dyspnea. He had had a moderately severe productive cough for many years.

He was acutely ill, dyspneic, orthopneic, and cyanotic. The heart was enlarged and the sounds were of poor quality. Gallop rhythm was present. There was a diminution in breath sounds and tactile fremitus over the left lower lobe. The pain continued for several days and then subsided. The cough was moderately severe and productive of several ounces of nonfoul mucopurulent sputum daily. The temperature varied irregularly between 100 and 101° F. A course of sulfadiazine was without effect on the clinical state. Repeated electrocardiograms were bizarre and suggested recent infarctions of the anterior and posterior surfaces of the heart. Because of a persistence and increase in the signs of fluid in the pleural cavity, roentgenographic examination was first performed ten days after admission. It revealed a localized pleural collection of fluid in the lower two-thirds of the left side of the chest. Aspirations of the chest on the eleventh, thirteenth, and fifteenth days after admission revealed large quantities of clear, nonfoul fluid (sympathetic effusion). The following day thick, green, foul pus was obtained by aspiration in the eighth intercostal space in the midscapular line.

Operation was performed at once, 300 c.c. of foul pus being evacuated from the pleural cavity. The walls of the empyema cavity were markedly thickened. Neither a lung abscess nor a bronchial fistula was observed.

During the remaining three weeks, the patient had an irregular temperature as high as 102° F. He continued to cough moderately, but the sputum was not foul. The operative wound became clean in several days and the foul odor disappeared. Shortly before death, signs of a pneumonia in the right lower lobe were observed.

Autopsy examination revealed cylindrical, nonputrid bronchiectasis and pulmonary fibrosis in the left lower lobe, nonputrid bronchopneumonia in the right lower lobe, pulmonary emphysema and coronary artery sclerosis with scattered myocardial fibrosis but no evidence of a myocardial infarct. The putrid pleural disease was no longer present.

This case illustrates the dangers of an undiagnosed putrid empyema. Roentgenographic examinations and pleural aspirations earlier in the course might have facilitated prompt diagnosis. Earlier operation might have prevented the fatality. The clinical history was confused by the bronchiectasis, severe cough, and copious sputum, all of which were essentially unrelated to the putrid empyema.

SUMMARY

The diagnosis of putrid empyema in the absence of foul sputum or other evidence of a pulmonary abscess is difficult. The lesion should be suspected and efforts at diagnosis persisted in when a given set of clinical manifestations are present. Prolonged delay in diagnosis may be fatal. The variable clinical manifestations are described. They consist primarily of outstanding and persistent chest pain, slight cough and expectoration, and an illness which is severe at least in the initial stage. Cough may be absent; the course may be chronic. The discovery of foul pus by aspiration of the pleura is the sole method of establishing the diagnosis. This disclosure will often be a surprise. Wide drainage of the main lesion and its ramifications will effect a cure unless the diagnosis has been delayed too long. Although a pulmonary abscess is not demonstrable, we believe, for reasons which have been advanced, that a putrid pulmonary abscess is the precursor of "surprise" putrid empyema. Résumés are presented of 3 of the 15 cases on which the paper is based.

PULMONARY EMBOLISM IN FRACTURES OF THE HIP

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CONSIDERABLE effort has been expended upon the mechanical correction of the osseous deformity in fractures of the hip for many generations, but little study has been given to the actual cause of the complications following this injury. Inadequate investigation is responsible for the marked discrepancy in the mortality figures from different institutions and in the clinical impressions of the causes of death. Therefore, we undertook a careful analysis of our own cases that came to autopsy in order to evolve, after a better understanding of the *modus operandi* of death, some means to reduce our mortality rate.

At the Queens General Hospital, 304 patients with fractures of the hip were treated from 1935 to 1944, with 86 deaths, a mortality of 28 per cent. Two-thirds of the deaths occurred in the group between 70 and 80 years of age. Findings in 25 autopsies were exceedingly illuminating. Investigation of the deaths revealed that 9 cases were due to pulmonary embolizations, 8 to bronchopneumonia, 3 to arteriosclerotic heart disease, 1 to hemorrhage from a duodenal ulcer, 1 to sepsis from a wound infection, 1 to sepsis from a severe purulent prostatitis, 1 to pyelonephritis, and 1 to voluntary starvation in a demented woman. Thus, 68 per cent of the necropsies exhibited pulmonary complications; of which 53 per cent were proved pulmonary emboli. The remaining 47 per cent were reported as bronchopneumonia; many of these cases were, possibly, the result of minute embolizations with secondary pneumonitis.

The high incidence of pulmonary embolizations prompted us to undertake a study of the factors that influence the pathogenesis. Venous thrombosis in the lower extremities has been established as the primary source of pulmonary emboli by Roessle,[1] Frykholm,[2] Bauer,[3] and other investigators. The sites of venous thrombosis in their relative order of frequency, as described by Frykholm,[2] occur in the veins of the calf muscles, the posterior tibial, the peroneal, the popliteal, and the veins of the adductor muscles, because damage to the intima of the vein is greatest in these regions. He stated, that under normal conditions, erect posture results in the distention of the lower extremity veins by the column of blood that they support. These veins become collapsed when the horizontal position is assumed. When a patient is confined to bed, the collapse of the calf vein is aggravated as the result of a compression force exerted against it by the resistance of the underlying mattress. In this manner, intima is pressed against intima. Continued pressure causes damage to the endothelial cells, because the nourishment provided to them by the sluggish stream of blood is inadequate. The injured cells liberate a thromboplastic substance which favors local thrombosis. The most frequent site of thrombus formation occurs at the valve structures because the lumen of the vessels is narrowest at these points, and the valves, acting as weirs, produce an eddy formation with the deposition of platelets.

An individual who suffers a fracture of his hip presents ideal conditions for the development of venous thrombi in the lower extremities. The blood

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TABLE I. PRESENT SERIES

Total patients treated		304
Total deaths		86
Total autopsies		25
Pulmonary complications	16	
Nonpulmonary complications	9	

stream is slowed by the complete voluntary immobilization of the extremities to avoid painful movements at the fracture site, by the associated reflex vasospasm induced by the fracture, and by the impaired cardiac circulation and the inadequate peripheral circulation of this age group. Concomitantly, changes in the chemical composition of the blood conducive to thrombosis formation result from the liberation of tissue ferments by the lacerated muscles, ligaments, and bone at the fracture sites. Intimal damage occurring when the patient is bedridden is

TABLE II. RÉSUMÉ OF TWENTY-FIVE AUTOPSIED CASES STUDIED

CASE NO.	PATHOLOGIC DIAGNOSIS	SITE OF FRACTURE	AGE (YR.)	TYPE OF TREATMENT	AMBULANT STATUS OBTAINED	TREATMENT STARTED DAYS AFTER ADMISSION	ILLNESS STARTED DAYS AFTER TREATMENT	TOTAL NUMBER DAYS IN HOSPITAL
1	Pulmonary embolism	Intertrochanteric	80	Smith-Petersen nail	No	4	1	14
2	Pulmonary embolism	Neck	80	Wilkie boots	Yes	11	1	42
3	Pulmonary embolism	Subtrochanteric	80	Sandbags	No	1	27	27
4*	Pulmonary embolism	Intertrochanteric	78	Smith-Petersen nail	No	7	1	8
5	Pulmonary embolism	Intertrochanteric	72	Russel traction	No	1	25	70
6	Pulmonary embolism	Neck	70	Smith-Petersen nail	No	9	Immediate	9
7	Pulmonary embolism	Intertrochanteric	70	Wilkie boots	No	1	11	20
8	Pulmonary embolism	Intertrochanteric	65	Wilkie boots	No	1	5	6
9	Pulmonary embolism	Subtrochanteric	54	Russel traction	No	14	3	16
10	Bronchopneumonia	Intertrochanteric	88	Russel traction	No	10	8	18
11	Bronchopneumonia	Neck	79	Sandbags	No	1	4	4
12	Bronchopneumonia	Intertrochanteric	78	Wilkie boots	No	1	11	14
13	Bronchopneumonia	Intertrochanteric	76	Wilkie boots	Yes	10	46	58
14	Bronchopneumonia	Intertrochanteric	75	Sandbags	No	1	1	8
15	Bronchopneumonia	Subtrochanteric	67	Russel traction	No	1	8	10
16	Bronchopneumonia	Intertrochanteric	65	Wilkie boots	Yes	7	10	20
17	Arteriosclerotic heart disease	Intertrochanteric	63	Sandbags	No	1	1	2
18	Arteriosclerotic heart disease	Subtrochanteric	63	Russel traction	No	1	1	1
19	Arteriosclerotic heart disease	Neck	60	Sandbags	No	1	16	18
20	Arteriosclerotic heart disease	Intertrochanteric	55	Russel traction, body spica	No	1	39	50
21	Sepsis, wound infection tion	Intertrochanteric	79	Smith-Petersen nail	No	2	6	31
22	Sepsis, purulent prostatitis	Intertrochanteric	73	Sandbags	No	1	2	6
23	Sepsis, pyelonephritis	Intertrochanteric	61	Wilkie boots	No	2	4	10
24	Hemorrhage, duodenal ulcer	Intertrochanteric	72	Russel traction	No	1	30	37
25	Starvation, voluntary	Neck	78	Russel traction	No	1	1	78

*Concomitant basilar artery thrombosis.

enhanced when spicas or Wilkie boots are utilized because of the greater pressure exerted against the calf veins by these plaster of Paris encasements.

The most dangerous type is the silent propagating thrombus, described by Ochsner[4] under the term "phlebothrombosis," because due to the lack of local symptoms, no precautions are undertaken. This insidious silent propagating venous thrombus was predominant in our series; not one patient gave any symptoms referable to the presence of venous thrombosis in the lower extremity. As a rule, some effort motion discharged the fatal pulmonary embolus; in one case manipulative reduction in the operating room; in another, transfer of the patient from a bed to wheel chair; in others, turning the patient from one side to the other in bed. Deaths occurred as early as the sixth day and as late as the seventieth day following injury.

The pathologic anatomy and the clinical course of pulmonary embolizations depend upon the size and the sterility of the embolus. Four of the nine fatal cases of pulmonary embolization resulted from massive emboli to the pulmonary conus, the pulmonary artery, or one of its branches. The patient was described as seized suddenly with severe pain, dyspnea, and cyanosis. A state of shock occurred rapidly, and death ensued in a few to thirty minutes. Pathologic examination of the lungs revealed little gross change, because death had been too rapid.

TABLE III. CLINICOPATHOLOGIC STUDY (CASES OF PULMONALY EMBOLISM)

CASE NO.	NUMBER OF ATTACKS	TYPE OF DEATH	TYPE OF EMBOLUS	PULMONARY PATHOLOGY
9	One	Sudden	Massive	Normal
8	One	Sudden	Massive	Normal
6	One	Sudden	Massive	Normal
3	One	Sudden	Massive	Normal
2	Multiple	Slow decline	Bilateral small	Bilateral infarcts
7	Multiple	Slow decline	Bilateral small	Bilateral infarcts
5	Multiple	Slow decline	Bilateral infected	Bilateral infected
1	None	Slow decline	Bilateral minute infected	Multiple small abscesses
4*	None	Sudden	Bilateral minute	Bronchopneumonia atelectasis

*Concomitant basilar artery thrombosis.

Three of the nine cases had clinical evidence of previous attacks of pulmonary embolization. Episodes of chest pain, dyspnea, and cyanosis occurred, which disappeared spontaneously after a few days. The patient was left with a residual weakness; this weakness is apparently a most important sign. At autopsy, emboli to the smaller branches were demonstrated which were associated with the formation of infarcts of the lung. The relative age of each infarction was determined by studying its structure and that of the corresponding adherent pulmonary embolus.

Two of the nine cases showed multiple minute emboli which occluded some of the smaller radicles of the pulmonary artery. These embolizations were not evidenced by any clinical signs or symptoms. Multiple embolic abscesses of the lungs and resultant sepsis were present in one case. A bilateral bronchopneumonia attended the other; the immediate cause of death in this case was a basilar artery thrombosis. The cases may be looked upon as incidental pulmonary emboli. The thrombi are too small, and the collateral circulation is sufficiently adequate to prevent an infarction. Small emboli become significant only when they are septic from infected veins, and produce multiple lung abscesses.

Sudden death from pulmonary embolism has been studied by Horn, Dack, Friedberg[5] and others; they found that one of two patterns may result. Complete or almost complete obstruction of both pulmonary artery branches, or the pulmonary conus due to a massive embolus, is fatal within a few minutes. Death results from asphyxia; the entire circulation of blood in the pulmonary artery is occluded. The right side of the heart in a few cases is dilated and the peripheral veins and organs show congestion. When emboli are too small to produce complete occlusion of the pulmonary artery, a superimposed spasm may complete the obstruction. Solitary embolization to either of the branches of the pulmonary artery may precipitate death by stimulating afferent vagal fibers with resultant vago-vagal reflex spasm of the coronary arteries and the bronchi, with severe cardiac inhibition.

Clinically, the diagnosis of the true cause of death was made in but one case of the nine patients that succumbed to pulmonary embolization, and this was entertained because of the sudden death of the patient. In all of the others, the clinical impression varied from bronchopneumonia to heart disease. This large diagnostic error may be due to the inability of the physician to examine the patient properly for Homans' dorsiflexion sign, tenderness of the calf muscles, cyanosis of the skin, and edema of the extremities. Particularly is this true when the plaster of Paris cast masks all local manifestations. On the other hand, physicians may not be cognizant that thrombosis of the calf veins is a frequent complication of fractures of the hip and, therefore, are not sufficiently vigilant.

Adequate efficient therapy for the prevention of embolism must be the desideratum if the mortality rates are to be lowered. Venous thrombosis occurs rarely when manipulative operative procedures are instituted early. These methods encourage ambulatory care of the patient, and the stasis of blood in the lower extremities and the pressure upon the calf region are to a large extent eliminated. Many of the patients in our series, in whom fatal embolization occurred, were confined to bed for a period of four to twelve days before any operative or manipulative procedure was done. This period of procrastination allows for venous thrombosis in the lower extremities. In fact, one case of massive embolization occurred while a reduction of the hip was being effected on the ninth postadmission day. Therapy included nailing in three cases, Russell traction in two cases, Wilkie or well-leg traction in three cases, and sandbag immobilization in one case. Eighty-eight per cent of the patients were bedridden not through choice, but because conditions beyond the control of the surgeon precluded mobilization of the patient. Thrombosis and embolism occurred in such cases because the patient did not actually obtain ambulatory status despite operative treatment to effect it.

Recent literature has been much concerned with the use of anticoagulation therapy in the form of dicumarol and heparin. Under such treatment, thrombus formation in veins can be prevented, and in the case of venous thrombosis, propagation of the original thrombus limited. Thrombosis and fatal embolization may occur after the drug has been discontinued. Since the treatment of fractures of the hip necessitates prolonged hospitalization and immobilization, a course of anticoagulation therapy must be continued over a period of months. This form of treatment not only becomes expensive, but also detrimental to the morale of the patient since it requires constant vigilance and venepuncture.

Venograms or phlebograms as a diagnostic method to discover venous thrombosis at its inception is not applicable to fractures of the femur. In those

cases where application of plaster of Paris casts are used in the form of a spica or Wilkie boot, x-ray will not demonstrate the passage of the opaque dye in the veins of the leg and foot because of the overlying opacity. Allen and associates[6] have produced data which seems to establish the unreliability of a phlebogram, since they have demonstrated positive clinical evidence of a thrombus with a negative phlebogram in one-third of the cases they studied.

To decrease the incidence of massive pulmonary emboli, early prophylactic venous ligation should be effected in patients who cannot have early operative and ambulatory treatment. Our mortality and morbidity may be diminished since many cases of pneumonitis may be the result of small pulmonary embolizations. Widespread interest has followed Homans'[7] contributions to the procedure of ligation of the femoral vein as a prophylactic measure for pulmonary embolization in cases of venous thrombosis of the lower extremity veins. Allen and co-workers[6] ligated in 202 patients up to January, 1943, with no mortalities; 60 per cent of his patients were over 50 years of age. They reported no occurrence of fatal embolization following ligation. They have demonstrated that the earlier the vein is ligated, the smaller the clot found and the less swelling of the leg results. Perhaps ligation of a normal femoral vein prophylactically may avoid edema in the lower extremities.

Venous ligation is not an emergency procedure. Ligation should be done within the first three days after the accident, as soon as shock is completely overcome. The site of election is the superficial femoral vein. Allen and associates[6] have demonstrated the necessity for bilateral venous ligation, because more often than not thrombosis may occur in the deep veins of both lower extremities. Following the ligations, the lumbar sympathetic block described by Leriche and Kunlin[13] and popularized by Ochsner and DeBakey[14] would overcome the vasospasm, enhance the collateral venous circulation, and minimize the incidence of edema. Then, the treatment of the fractured hip may be instituted at any time convenient for the correction of the disability without the fear of pulmonary embolization.

CONCLUSIONS

1. Fatal pulmonary embolization from venous thrombosis in the veins of the lower extremities is the most frequent cause of death in cases of fracture of the hip.

2. Venous thrombosis and embolism are less frequent in patients who are made ambulant early.

3. Prophylactic bilateral superficial femoral vein ligation combined with lumbar sympathetic block is suggested as most ideal for the prevention of pulmonary embolism in fractures of the hip; especially, in those who cannot be made ambulant early.

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THE TREATMENT OF PROGRESSIVE BACTERIAL SYNERGISTIC GANGRENE WITH PENICILLIN

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INTRODUCTION

PROGRESSIVE bacterial synergistic gangrene is now accepted as a clinical entity. Its underlying causes are known, its mode of onset is understood, its bacterial synergistic etiology has been repeatedly confirmed, and its characteristic appearance after it has become established should be unmistakable. Cullen,[1] in 1924, was the first to give a comprehensive case report of this condition, although at least one authentic case had appeared in the literature before that time.[2] Since Cullen's publication, there have been almost 100 reports of typical cases. Stewart-Wallace[3] gave an excellent review with bibliography of thirty-seven cases up to 1935 and Dodd, Heekes, and Geiser[4] continued the story, totaling eighty-six cases up to 1939. Not included in their review, there have been summaries or case reports by Pergola and Rosenfeld[5] in 1938, by Hulten,[6] by Ducrey,[7] and by Touraine and Duperrat[8] in 1939. Later, reports appeared by Antonioli,[9] by Brodie and Bouck,[10] by Constantinescu and Vasiliu,[11] by Mester,[12] by Vier[13] in 1940, by Lichtenstein[14] in 1941, by Gurruchaga and Manzoni,[15] by Paulino,[16] by Vara-Lopez[17] in 1942, by Neary and Rankine[18] in 1943, by Davison, Sarnat, and Lampert,[19] and by Leonard[20] in 1944.

While some of the authors quoted in these reviews confused this disease with the chronic undermining, burrowing (nongangrenous) ulcer, the great majority have presented authentic cases. Undoubtedly many other cases have been observed and have not been included or have not been reported. One of us (F. L. M.) has reported one such case not included, besides the five mentioned in the list,[21] and has seen and studied eight others which have not been reported at all, in addition to the three cases which are the subject of this paper—seventeen in all, and in every one of these cases the essential organisms have been found. Obviously it is not as rare a disease as was once thought and it should be recognized much more readily than it is.

One of us (F. L. M.), in 1926, found the essential bacterial agent to be a microaerophilic nonhemolytic streptococcus which was present in pure culture in

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the advancing periphery of the lesion in one of the cases described by Brewer and one of us (F. L. M.)[22] This organism was associated with a hemolytic *Staphylococcus aureus* in the gangrenous margin. The combination of the streptococcus and the staphylococcus was shown to produce in animals a gangrenous lesion simulating to a degree the process in human beings. This lesion could only be obtained when the organisms were combined and not when they were inoculated in pure culture, thus demonstrating their synergistic action. This synergistic action was clearly confirmed in a later case, thus justifying the name "progressive bacterial synergistic gangrene."[23] Those who doubt this etiology have given altogether too much credit to those bacteriologic reports which included *no* anaerobic technique, for the microaerophilic nonhemolytic streptococcus can be found only by careful anaerobic methods. It forms a tiny colony on the anaerobic plates and is readily overgrown by such organisms as *Bacillus proteus, Escherichia coli,* or staphylococci.[24] It is hard to understand why anaerobic procedures are so often neglected in the analysis of surgical infections. Any chronic infection is subject to secondary contamination so that many organisms have been found associated with this lesion, but the consistent finding of the microaerophilic nonhemolytic streptococcus in pure culture in the spreading periphery of the lesion almost certainly establishes it as the essential causative organism. In the necrotic and the necrobiotic zones, however, it is invariably associated with staphylococci and frequently with other organisms as well.

The development of the lesion follows a definite course. It usually begins around tension sutures placed in a wound of the abdominal wall or chest which has been made for the purpose of draining either an intraperitoneal or intrapleural abscess. Frequently the intrapleural infection has developed secondary to a lung abscess of the putrid variety. The skin around the tension suture first becomes red, swollen, and tender and takes on a dusky appearance resembling a carbuncle with a slowly developing necrosis of the skin. This frequently occurs about ten to twenty days after the wound has been made. Removal of the tension sutures at that time does not control the infection which steadily spreads in all directions from the central point and gradually produces an ulcer. The lesion is usually exquisitely tender and also painful and not infrequently the entire disposition of the patient changes as the infection progresses, in spite of all the doctor can do for it.

As the ulcer spreads, the destruction of tissue is seen to be very superficial. The gangrenous skin dissolves away at its inner margin which is slightly undermined, but at the periphery of the gangrenous zone it is intimately adherent to the surrounding skin which, however, has a dusky appearance of impending death and is raised from one-half to one centimeter above the normal surface of the skin. The inner margin of this raised zone is dark purple in color and has an irregular crenated appearance. Beyond the raised, swollen area, there is a zone of erythema which may vary in width from 5 or 6 mm. to 4 or 5 cm., the outer margin gradually fading off into normal skin.

If early treatment is not instituted and the ulcer continues to grow, granulations develop in the center, and residual islands of epithelium may form a thin epithelial surface. As time goes on, the progress gradually slows down but it does not stop. It goes on inexorably in spite of any local application which the ingenious surgeon may devise. Frequently surgeons have attempted to trim away the dead tissue but almost invariably, in the past, without a radical removal of the whole lesion well beyond the zone of erythema, there has been a recurrence of the infection and a redevelopment of the characteristic marginal zones. In the past, the only cure for this condition has been com-

plete and wide excision. Then a number of different antiseptics have been applied to minimize the chances of a recurrence. Zinc peroxide has been the most effective we have tried in preventing recurrence after the excision because the microaerophilic nonhemolytic streptococcus is susceptible to it and that is the organism which has to be stopped if the lesion is to be cured. But without excision, neither zinc peroxide nor any other external application has been able to halt the steady advance of the organisms which we know to be active in the periphery of the lesion, and heretofore no medication has been able to halt their advance from within. The sulfonamides have not been effective in controlling this condition, but with the advent of penicillin, a new agent has been found which may not only prevent a recurrence after the excision, but may obviate the necessity for excision by halting the progress of the lesion and permitting a spontaneous separation of the slough. This has been demonstrated very strikingly in two patients whom we have recently treated and whose case records are reported herewith. In a third patient with multiple lesions, although it was of definite benefit, it was not quite equal to the situation, probably because the penicillin was inactivated by secondary contaminants.

CASE REPORTS

CASE 1 (Jewish Memorial Hospital No. 35114 and 40989).—N. W., aged 67 years, was admitted Feb. 15, 1944, and discharged June 25, 1944 (under the care of S. T. F.).

History.—One week prior to admission, the patient was suddenly seized with a chill, a fever of 103° F., and a cough with yellowish sputum. On the day of admission, he complained of pain in the left side of the chest.

Physical Examination.—The patient was an emaciated, old man who appeared acutely ill. There were dullness, bronchial breathing, and râles over the left lower lobe. The heart sounds were muffled and distant. X-ray films revealed an irregular bronchopneumonia throughout the left lower lobe with signs of old tuberculosis in the left apex and hilus. There was a small amount of fluid in the costophrenic angle.

Course.—Sulfadiazine was begun with a dosage of one gram every four hours and an equivalent amount of sodium bicarbonate. There was no apparent response to chemotherapy and the sulfadiazine was discontinued at the end of one week when hematuria appeared. Gradually a breakdown of lung tissue occurred in the upper part of the left lower lobe (see Fig. 1). The sputum became profuse but not foul smelling. By the end of the fifth week, the pleural effusion had increased considerably and several fluid levels were seen indicating localized pockets containing gas. Expectoration then brought forth greenish-yellow, foul-smelling pus.

A rib was resected on the forty-fifth day of the disease and several ounces of putrid pus were removed. A rubber drainage tube was inserted. The temperature came down somewhat, but the patient's condition remained poor. Five days later a chest x-ray revealed a drained pleura and a retracted lung (Fig. 2). Seventeen days after operation, the edges of the wound gradually became swollen and inflamed and within two or three days the margins became necrotic. Necrosis continued to spread in spite of frequent cutting away of the dead tissue, and during the next two weeks a steadily progressive gangrenous ulcer developed around the drainage site. One month after operation and sixteen days after the infection of the chest wall, one of us (F. L. M.) was asked to see the patient. The appearance then was that shown in Fig. 3, which is typical of progressive bacterial synergistic gangrene.

The man was desperately ill and his death was expected from day to day. Any operative procedure would almost certainly have precipitated his death, even the simple excision of the ulcer. He was, therefore, placed on penicillin therapy with a dosage of 200,000 units a day, being given 25,000 units intramuscularly every three hours. Twice a day, 10,000 units were also instilled through a tube into the pleural cavity. A culture of the necrotic margin yielded a mixture of organisms including the microaerophilic, nonhemolytic streptococcus and a hemolytic *Staphylococcus aureus*, the combination of organisms which has been demonstrated in other cases to be the etiologic factor in progressive bacterial synergistic gangrene. There were also present an anerobic hemolytic streptococcus, and an aerobic nonhemolytic streptococcus, a *Clostridium welchii*, a member of the pseudomonas group, and *Bacillus subtilis*.

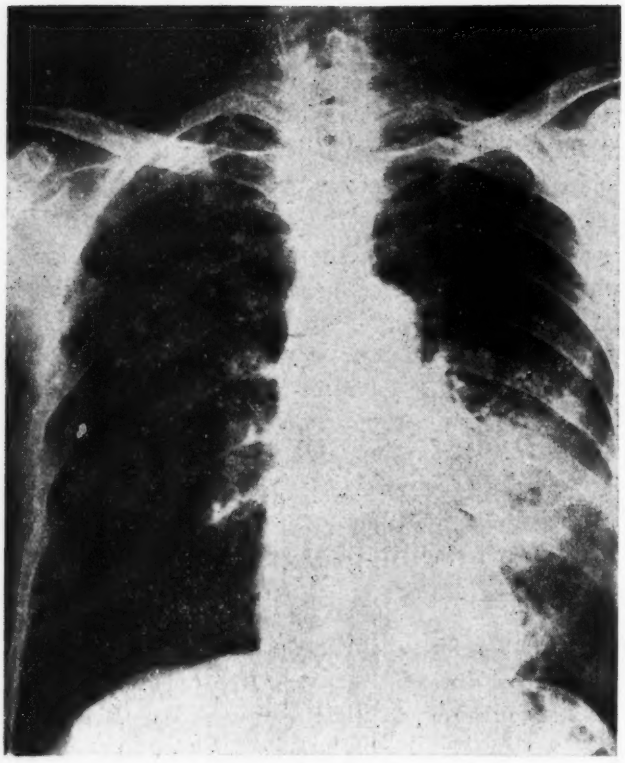

Fig. 1 (Case 1). Lung abscess fifteen days after the onset of symptoms.

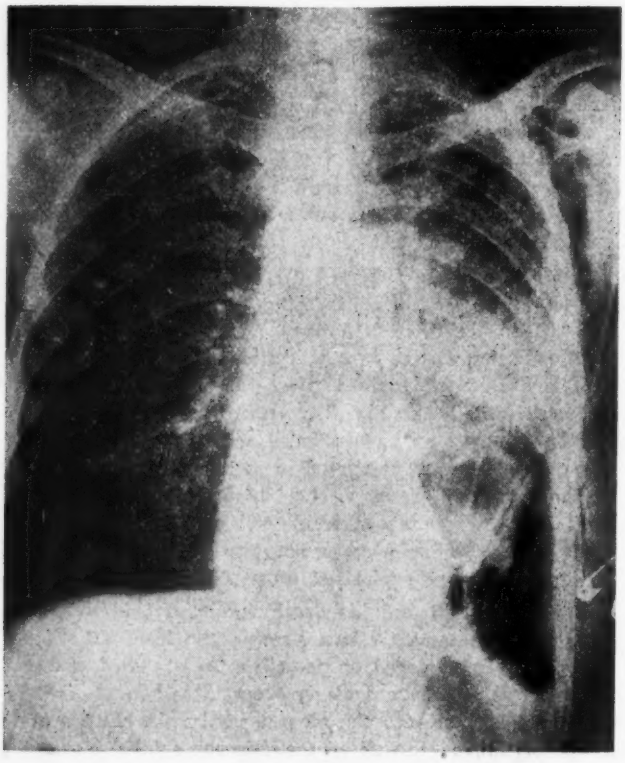

Fig. 2 (Case 1).—After drainage of putrid empyema, the lung with the abscess cavity has retracted upward; this is the fiftieth day of the disease.

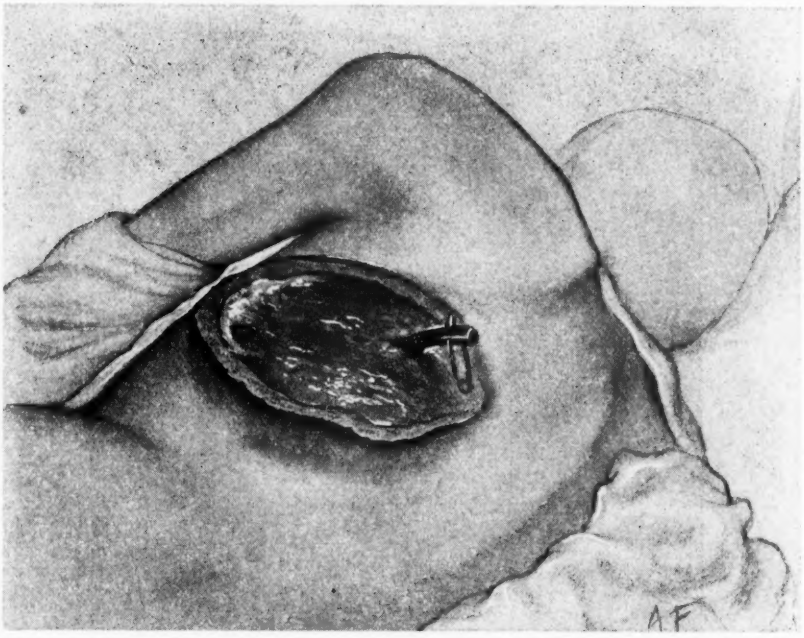

Fig. 3 (Case 1).—The lesion sixteen days after the development of the ulcer. This shows the drainage wound surrounded by a wide area of bright red granulations, a narrow zone of gangrene interrup'ed in two or three places around the circumference, firmly adherent to the raised necrobiotic zone and at the periphery, a zone of erythema varying in width from a few millimeters to about three centimeters.

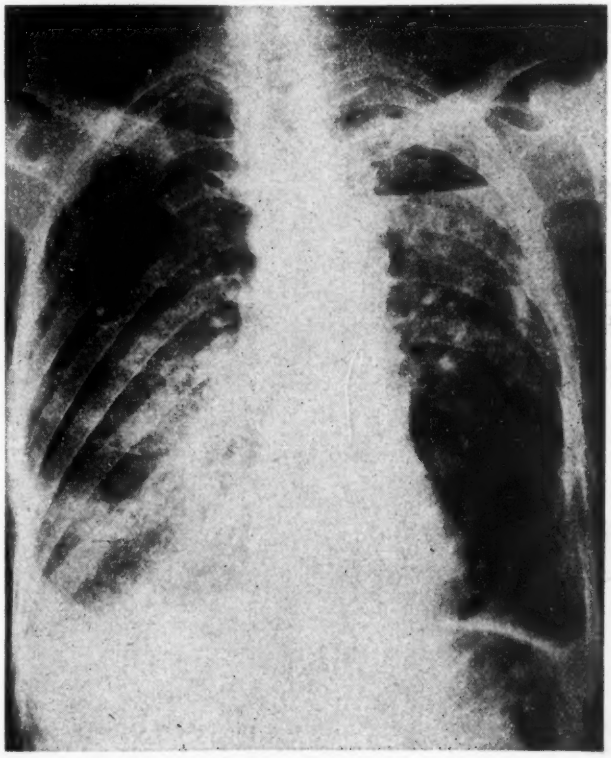

Fig. 4 (Case 1).—This is the one hundredth and sixth day of the disease; the infection has spread to the right lung and the fluid level is seen on the left side.

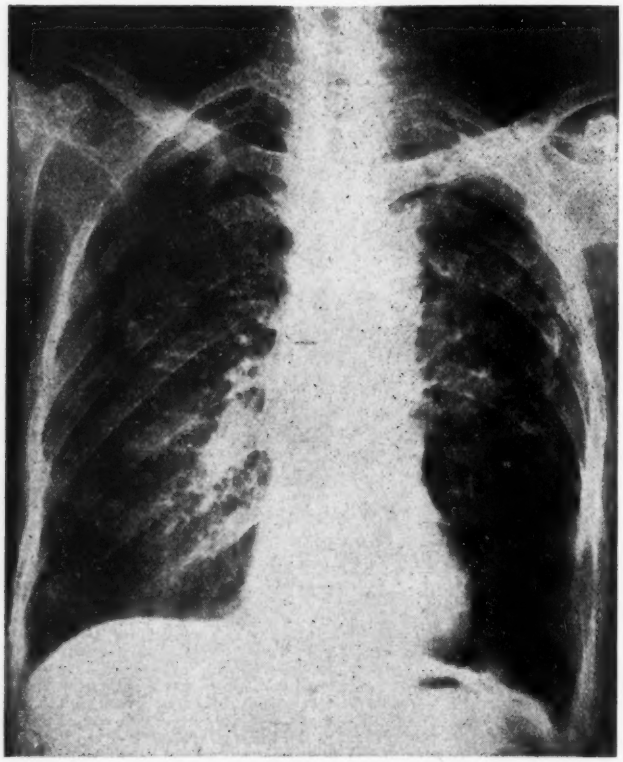

Fig. 5 (Case 1).—This is the one hundredth and twenty-fifth day of the disease; the lungs are clearing.

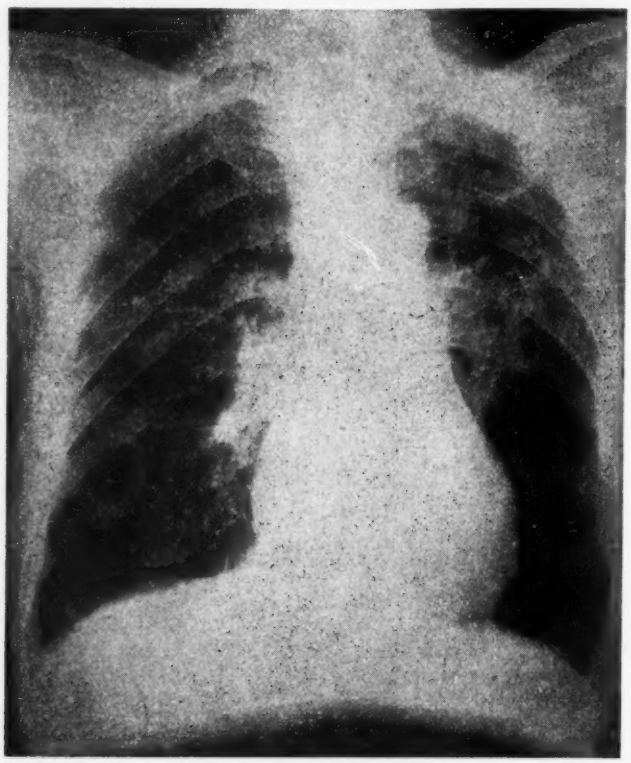

Fig. 6 (Case 1).—One year after the onset of the disease; the lungs show only a residual fibrosis.

Within one week, there was marked improvement and the progress of the ulcer was halted. The necrotic tissue gradually separated off. After ten days, the wound was clean and granulating with very little discharge from the pleural cavity. Furthermore, the process in the lung cleared, although multiple cavities were still evident within the left lower lobe. From that time on, his condition gradually improved. The cough and expectoration diminished and his appetite and strength increased. The pleural cavity closed. On the twenty-fifth day, the penicillin was reduced to 50,000 units daily. However, a few days later, x-rays revealed a fluid level in the upper left pleural cavity and a spread of infection to the lower right lobe (Fig. 4). The dose of penicillin was, therefore, raised to 100,000 units a day. The lung fields then gradually cleared and the patient was allowed up. Epithelium grew in rapidly from the margin of the ulcer, Pinch grafts were then applied to the ulcer and the defect was rapidly covered with epithelium. The lung lesions progressively and rapidly diminished and he left the hospital just two months after the beginning of the penicillin therapy (Fig. 5). He has remained well during the succeeding eight months (see Figs. 6 and 7).

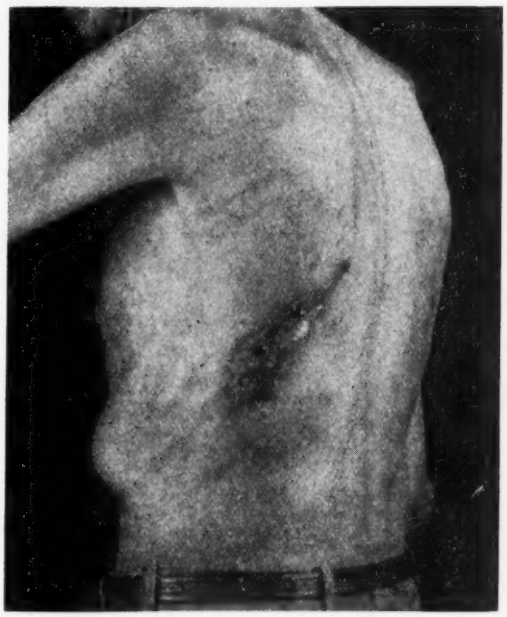

Fig. 7 (Case 1).—This shows the healed scar six months after wound healing.

This case illustrates the failure of sulfadiazine to prevent the development of a putrid lung abscess which later developed an associated progressive bacterial synergistic gangrene of the chest wall following the drainage of a putrid empyema. Although there were multiple lung abscesses, which were not drained surgically, the whole process came under control with penicillin. As far as we know, this is the first case of this kind on record in which the patient was treated successfully with penicillin.

CASE 2 (Presbyterian Hospital No. 744592).—G. H., aged 53 years, was admitted May 2, 1944, and discharged July 14, 1944 (under the care of H. D. H).

History.—Suddenly after breakfast one morning, without previous symptoms, the patient began to have pains in the midabdomen coming on in waves and growing gradually more and more severe. The pain was relieved somewhat by flexing the thighs. After a few hours, the pain gradually shifted to the lower abdomen with local tenderness in that region and pain across the lower back. There was no nausea, vomiting, or fever.

Physical Examination.—A mass was felt in the lower right side of the abdomen, which was quite tender, hard, and lobulated. The muscles of the abdominal wall over it were spastic. Elsewhere, the abdomen was quite soft and nontender.

Course.—An emergency operation was performed about nine hours after the onset of symptoms. The findings were quite unusual. There was an opening in the mesentery of the

terminal ileum about 50 cm. from the ileocecal valve. Through this opening, a loop of small intestine had herniated itself, running from left to right. The loop was about 25 cm. in length, swollen and obviously gangrenous. The entire terminal ileum had rotated on its mesentery including the herniated loop so that the intestine was gangrenous from a point about 2 cm. above the ileocecal valve upward for a distance of over 100 cm. The vessels in this portion of the mesentery were thrombosed. The gangrenous intestine was excised and an ileocecostomy was done. An enterostomy tube was then placed in the small intestine just proximal to the anastomosis, with a Witzel technique. The enterostomy tube was brought out at the lower end of the wound, which was then closed around it by means of interrupted sutures of No. 00 chromic catgut with a bolt suture of heavy silk taken from the skin on the right of the wound to the linea alba on the left as a tension suture. A culture from the peritoneal cavity preliminary to the resection showed no growth of organisms.

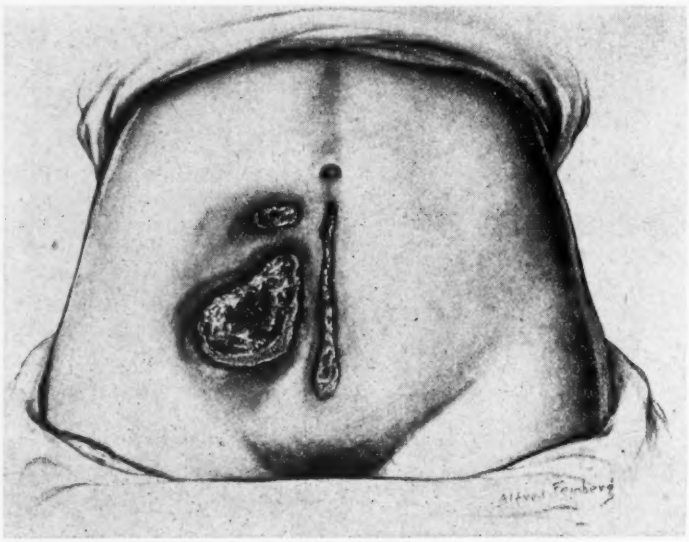

Fig. 8 (Case 2).—An artist's drawing of the lesion showing the central area of granulation tissue, a zone of suede leather gangrene around part of the margin, the raised purple zone, and the wide erythema. The small lesion above coalesced with the large lesion before penicillin was begun.

The patient was given sodium sulfadiazine, at first intravenously, and then sulfadiazine by mouth. On the fourth day, the sulfadiazine level had reached 18 mg. per cent. At that time there was a negative fluid balance with very little renal outflow and the urine showed sulfadiazine crystals and a few red blood cells. The sulfadiazine was stopped. Everything seemed to progress satisfactorily except for a moderate rise in temperature to 101.2° F. on the seventh day. The ileostomy tube was taken out on the eleventh day and the sutures were removed. However, around the bolt suture hole, an infection appeared on the fourteenth day and this gradually spread, appearing first as a raised dusky area of skin which steadily increased in area. On the twenty-second day, penicillin was introduced into the center of this area but it had very little effect. Gradually an ulcer formed and the whole region became extremely tender and painful. Her temperature gradually mounted, rising with moderate spikes from 101 to 102° F. and on the thirty-fifth day it was recognized as a typical progressive bacterial synergistic gangrenous ulcer with an inner zone of gangrene, a middle raised purple zone, and an outer erythematous zone. The whole lesion was approximately 8 by 10 cm. in diameter (Fig. 8). On the thirty-seventh day, penicillin was started intravenously in a dosage of 25,000 units every three hours. It was given continuously as an intravenous drip, 200,000 units being introduced in twenty-four hours. This treatment was followed by a striking improvement in the whole process. The spread of the infection ceased, the necrotic margin separated off spontaneously, and the whole area flattened down to the level of normal skin. The granulations took on a healthy appearance and new skin began to grow in from the margin. Penicillin was stopped on the forty-eighth day, after nine days of treatment.

Cultures from the lesion before the administration of penicillin revealed the essential organisms, a microaerophilic nonhemolytic streptococcus and a coagulase positive hemolytic *Staph. aureus*. There was also present a microaerophilic hemolytic streptococcus. On the

third day after stopping the penicillin, cultures of the wound showed no growth. On the twenty-seventh day after the start of penicillin, the patient consented to a skin graft. This might have been done fully two weeks earlier. Twenty small, deep, Davis skin grafts were planted from the thigh, all of which were successful and the wound was completely covered with epithelium ten days later (Fig. 9).

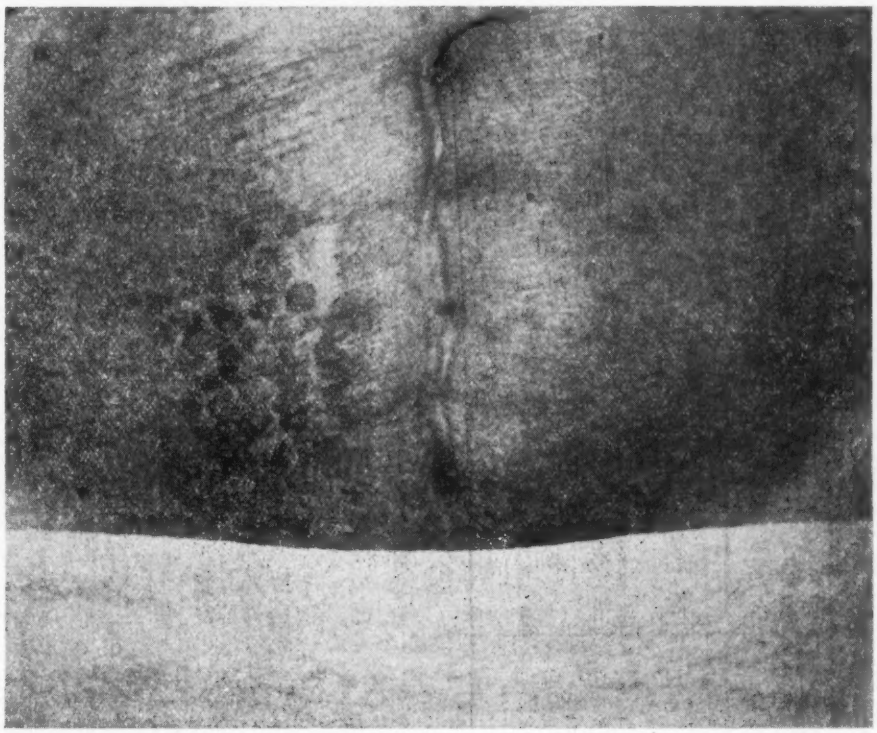

Fig. 9 (Case 2).—The healed scar nine months after wound healing.

CASE 3 (Presbyterian Hospital No. 763838.—B. B., aged 42 years, was admitted Nov. 9, 1944, and died Dec. 27, 1944 (under the care of F. L. M.).

History.—One and one-half years before admission, a boil developed on the lateral side of the right leg. It broke down and then formed an ulcer which gradually spread with a gangrenous margin, leaving in the center a granulating area on which epithelial islands appeared. Soon similar lesions developed on other parts of the body. They failed to respond to any treatment tried and the patient was finally admitted to a suburban hospital. Here he was given 550,000 units of penicillin over a period of three weeks. Penicillin was also applied locally but without benefit. Sulfadiazine and gentian violet were also tried without effect. He was then sent to New York to the Presbyterian Hospital on the dermatology service. At that time, there were five large and four small gangrenous ulcers on the body. Here a search was made for amebae but none were found either in the ulcers or in the stools. The Wassermann and Kline tests were negative. Wet dressings of potassium permanganate were applied without effect. The patient was then referred to the surgical service.

Physical Examination.—The patient was a well-developed, well-nourished man lying quietly in bed without much discomfort, but appearing chronically ill. There were twelve gangrenous ulcers on different parts of his body in varying stages of development. There was one on the right leg, one on the left thigh, one over the right hip, six on the anterior part of the chest, and three on the inner surface of the right arm, which had developed since admission to the hospital. These lesions, while they had most of the characteristics of the progressive bacterial synergistic gangrene as described in previous reports, differed in three respects. In the first place, they were multiple; second, they were not excruciatingly painful; and third, the zone of gangrene was relatively narrow (see Figs. 10 to 13).

They were all characterized by a raised, purple margin, irregular in outline and surrounded by a zone of erythema varying from ½ to 2 cm. Inside the raised purple zone, there was a gangrenous zone of varying width. In many places it was entirely absent and in other places it was as much as 1 cm. wide. The infection was apparently active in all of

the ulcers. There was a good deal of necrosis of the subcutaneous tissue with innumerable, small pockets in the subcutaneous fat and innumerable islands of granulation tissue, some of which looked healthy, while others appeared infected. In the central portion of the ulcers, there was a varying amount of epithelium which had evidently arisen from residual islands not destroyed by the infection. This was most marked in the oldest lesion which was on one leg, while there was only a small area of new epithelium on the lesion over the hip and the most recent lesions on the arm.

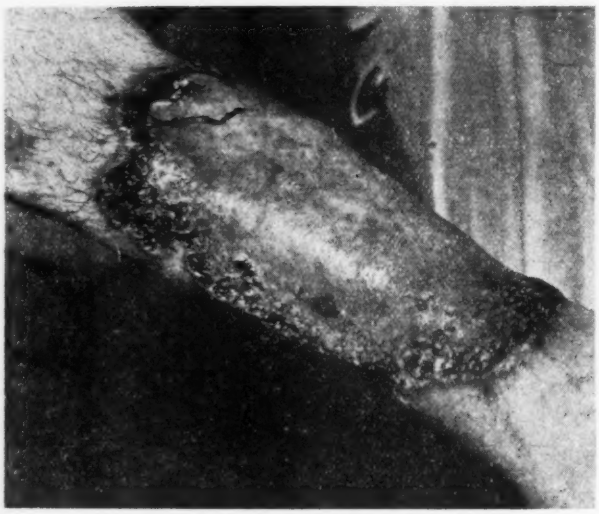

Fig. 10.

Fig. 11.

Fig. 10 (Case 3).—Lesion on the leg where the disease started one and one-half years before, showing a wide central area covered with thin regenerated epithelium, a spreading margin with only a narrow shread of gangrenous skin around part of the margin, a raised purple zone, and a narrow zone of erythema.

Fig. 11 (Case 3).—Two large, old areas on the chest showing narrow gangrenous zones and considerable areas of regenerated epithelium which have become secondarily broken down by the infection. The two smaller areas below the clavicles show more typical suede leather gangrene, raised purple zone, and wide erythema. Two small areas in the early stages are seen on the lower right side and two more on the inner side of the arm.

Fig. 12.

Fig. 13.

Fig. 12 (Case 3).—One of the older lesions above the left knee with narrow gangrenous and raised purple zones and a considerable area of regenerated epithelium.

Fig. 13 (Case 3).—One of the more recent lesions on the right hip and flank showing a wider zone of suede leather gangrene, a raised purple zone of varying width, and a very small area of regenerating epithelium.

Cultures from these ulcers showed a multitudinous number of bacterial species including the microaerophilic nonhemolytic streptococcus and the *Staph. aureus*, the essential organisms in the production of this disease. There were also *E. coli* and *Pseudomonas pyocyanea*, aerobic nonhemolytic streptococci, diphtheroids, and microaerophilic hemolytic streptococcus.

Course.—Patients in two previous similar cases having responded well to large doses of penicillin without surgery, the patient was given 25,000 units every three hours intramuscularly, but there was very little improvement after a course of two million units in ten days. The ulcers became cleaner, but the margins advanced, particularly the most recent lesions. It was decided, therefore, to excise the ulcers. When that decision was made, the penicillin was stopped and during the next four days the exudate from the ulcers considerably increased. Penicillin was, therefore, tried again for ten days but again the improvement was not sufficient to warrant continuing it. All but one of the lesions were, therefore, excised and three different kinds of local treatment were tried—penicillin, streptothricin, and zinc peroxide, each in a carbowax base. One ulcer was left as a control. All three of these forms of treatment seemed to have control over the infection but streptothricin produced the cleanest wound.

Suddenly early in the morning of the seventh postoperative day, the patient went into profound shock and died. Autopsy revealed a large pulmonary embolus, apparently coming from the iliac veins.

DISCUSSION

The first case illustrates a typical chest wall lesion resulting from the drainage of a putrid empyema arising from a lung abscess. The patient's condition was extremely critical and it seemed to those who were taking care of him that the outlook was hopeless. There can be no question about the efficacy of penicillin in this case, even though the bacteriologic examination showed a mixture of organisms. The extensive lung involvement must have been the chief factor in his downward course before the administration of penicillin and this became reactive when the dose of penicillin was reduced, coming under control again when the dose was increased. This seems to be added evidence that penicillin was effective in controlling the lung condition as well as the ulcer. At one time, we thought surgery would be necessary, but steady improvement both in the clinical course and in the x-ray findings obviated the necessity for surgery in the lung as well as in the chest wall.

The second case illustrates the abdominal wall form of this disease, the organisms almost certainly coming from the intestinal tract and establishing themselves in the abdominal wall where the circulation was compromised by the pressure or tension of the bolt suture. In the great majority of cases, this element of pressure by a suture can be demonstrated and indicates not only the necessity for a synergistic action by bacteria, but a third factor which lowers the resistance of the tissues, permitting the establishment of an organism not primarily pathogenic. In this case, the suture was carried deeply into the muscles of the abdominal wall and infection arose in the incision as well as around the suture. Furthermore, another organism, namely the hemolytic microaerophilic streptococcus, was also present and certain features of undermining seemed to indicate that we had here a combination of the synergistic gangrene and the undermining ulcer. However, the undermining, burrowing ulcers, in our experience, have not responded to penicillin and it does not seem likely that this was a major factor in the infection because of the prompt response of the lesion in this case to penicillin. Again surgical excision, which has been essential in the cure of this condition in the past, was obviated. Furthermore, the promptness of the response to penicillin leaves no doubt about the efficacy of the drug in this case. The patient tolerated the intravenous administration of penicillin and greatly preferred it to the periodic intramuscular injections which were extremely painful.

The third case falls into the group not associated with either the intestinal tract or the lungs, but the group in which the organism probably gets in as a secondary contaminant into an area compromised by the previous activity of the staphylococcus. Once established, however, the microaerophilic nonhemolytic streptococcus is the one which leads the advance into the periphery and prepares the ground for the secondary spread of the staphylococcus. It is the latter organism which is chiefly responsible for the death of tissue in the gangrenous zone and in the raised purple necrobiotic zone. The infection was obviously very superficial, as indicated by the extensive regeneration of the epithelium in the center of the older lesions. Penicillin, in this case, cut down the amount of exudate and probably had some antibacterial action on certain of the bacterial species, but the presence of Gram-negative rods in all of the lesions prevented its control of the infection. We demonstrated in the laboratory that the *E. coli* and *Ps. pyocyanea* both elaborated penicillinase, which completely inactivated penicillin in vitro. It may very well be that if we could have controlled these Gram-negative rods by some other medication, infection even though very extensive might have come under control with the penicillin. There can be no doubt about the improvement during the period of penicillin administration, but it was felt that the wide extent of the process would require surgical removal of a great part of the diseased tissue. The patient stood the operation very well indeed, and the postoperative course was entirely satisfactory up to the time of the pulmonary embolus, which was a most unfortunate accident and thoroughly unexpected. We do not feel that failure in this case in any way detracts from the value of penicillin in the disease, but emphasizes the necessity for a complete bacteriologic analysis of such lesions and the control of all of the bacteriologic elements. The inactivating of penicillin by secondary contaminants certainly must be recognized as one of the important limitations to penicillin therapy.

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ILEOSTOMY AND COLECTOMY IN CHRONIC ULCERATIVE COLITIS

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REVIEW of the statistics on cases of nonspecific ulcerative colitis at the University of Minnesota Hospitals for the ten years ending Jan. 1, 1944, showed an 8 per cent mortality among those twenty-five patients treated surgically and a 28 per cent mortality among those fifty-seven confined to conservative types of management.[1] Since these findings indicate the importance of surgical care in these cases, it seemed worth while to try to make a thorough study of the most satisfactory surgical techniques and methods of postoperative care. This present emphasis on the surgical management is, of course, a temporary one to be employed only until the true nature of the disease shall have been discovered, when it is presumed that less radical measures may suffice.

The previous study pointed out that the safest procedure in those cases requiring any surgery at all is to perform ileostomy and to allow at least three months to pass before undertaking other procedures. The indications for ileostomy have been presented elswhere, and will be only mentioned here. Nonspecific ulcerative colitis can be classified into the fulminating cases, the moderately severe ones with remissions and exacerbations, and the very mild ones. These last cases, the very mild ones, can usually be handled medically. All others are candidates for ileostomy at the University of Minnesota Hospitals. Emergency indications are: uncontrollable hemorrhage, fulminating disease, impending perforation, and obstruction. Elective indications are: chronic ulcerative colitis resisting all forms of medical treatment, segmental ulcerative colitis, very early ulcerative colitis, and polyposis including those cases with possible malignancy.

This paper was to have been presented at the meeting of the Society of University Surgeons, New York, N. Y., Feb. 8, 1945. (Meeting canceled.)

The type of ileostomy now employed has developed gradually in the past few years. At first, all ileostomies were of the double-barreled type, the loop being brought out through the main incision. Most of these wounds healed nicely, but about one-third of them broke down under the influence of the unspent digestive ferments poured forth from the ileostomy. This has, in the past, been one of the great objections to ileostomy posed by internists and surgeons alike. Excoriation of the skin, which results from constant bathing in the unspent secretions coming from the bowel, has been a second factor. Finally, the progressive emaciation born of loss of appetite and seepage of plasma from painful raw surfaces has caused many clinicians to throw up their hands in horror at the suggestion of ileostomy in any but the most extreme cases.

These problems seem to have been largely overcome by the employment of the ultimate regimen indicated in the present report. It has become the impression of most members of the surgical and medical staffs at the University of Minnesota Hospitals that ileostomy is less a burden than any but the most mild cases of colitis. One patient with ileostomy-colectomy does well enough to play professional football and to have declined to have ileoproctostomy performed.

In the sixteen months after Jan. 1, 1944, twenty-three new cases of ulcerative colitis have come to the attention of the medical and surgical staffs at this hospital. One was localized and was resected by another member of the staff. Of the remaining twenty-two patients, only seven did not have surgery; thirteen had ileostomies as described. The remaining two had ileostomies by other methods by other operators; both developed fecal fistulas. Of the seven not receiving operation, two died, two improved, and three failed to improve.

This review of recent experience indicates the favor in which proper surgical management is held at the present time at this clinic. It has been learned by painful experience that the most extreme care and precision must be exercised with every detail of the preoperative, operative, and postoperative care of these cases, and with this finding in mind, the procedures employed are described.

ILEOSTOMY

Preoperative Preparation.—The bulk of the candidates for ileostomy have been troubled with profuse liquid stools many times a day, the frequency and debility of which have been only increased by administration of the high-protein, high-caloric diets ordinarily employed at Minnesota on poor-risk patients.[2] It has, therefore, been the practice to fortify these individuals for ileostomy by continuous intravenous infusion of from 2 to 3 liters of 20 per cent dextrose solution in distilled water during the twenty-four to thirty-six hours before surgery. This is usually supplemented with plasma, as the plasma protein is ordinarily depleted. Crude liver extract (Campalon*) and the available purified vitamins are added to the dextrose solution.

Preoperative preparation includes 100 mg. of pentobarbital the night before surgery, repeated in the morning if the patient is not drowsy, also morphine and hyoscine as in routine preparation for other procedures. Wangensteen nasal suction is instituted before the patient leaves the ward, and is maintained usually the next two days. Enemas are scrupulously avoided.

Operation.—

Anesthesia: The patient is made as comfortable as possible on the operating table, and an 8 cm. left lower rectus incision is made under ½ per cent metycaine local infiltration (Fig. 1). The rectus sheaths are not infiltrated with 1 per

*Winthrop Chemical Company, New York, N. Y.

cent solution until exposed, as a precaution against puncturing the bowel. The rectus muscle is retracted laterally, and the abdomen entered.

Choice of level: No manipulation of the colon is performed. The desired segment of bowel can almost regularly be easily identified by the presence on the antimesenteric border of the terminal 10 cm. of ileum of the ileocecal fold described by Kelly and Hurdon[3] (not mentioned in the standard anatomy atlases and texts). It is delivered into the field; the diameter of the bowel is measured and it is carefully inspected with a view to making the ileostomy definitely above the level of involvement.* The mesentery is infiltrated with metycaine as far posteriorly as possible, and is divided at the chosen point for from 8 to 10 cm. toward the root, tying the well-isolated vessels encountered with No. 0 chromic catgut (Fig. 2).

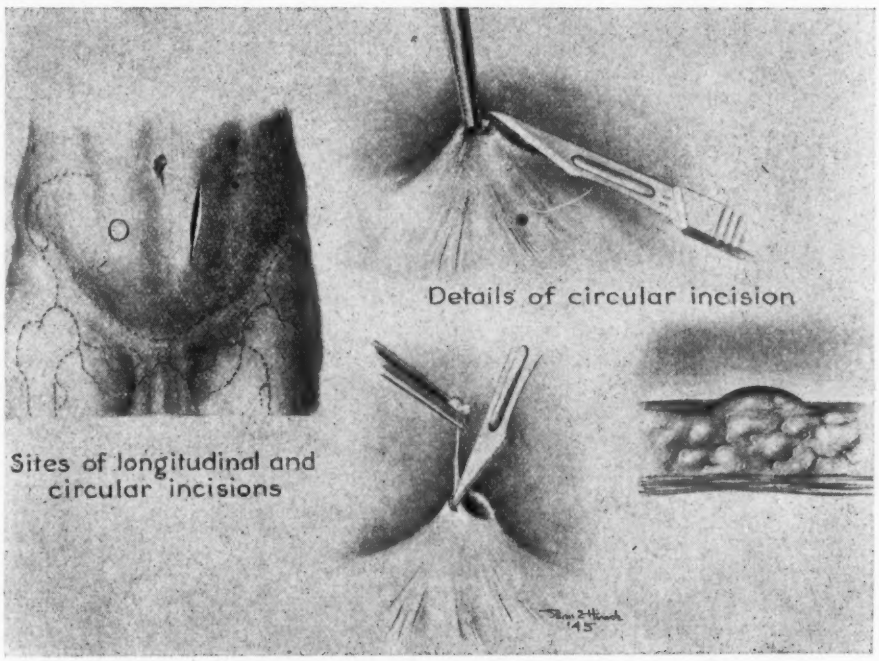


Fig. 1.—Details of the incisions employed for single-barreled terminal ileostomy. An 8 cm. left lower rectus incision is made. A circular skin incision two-thirds the diameter of the ileum to pass through it is made 6 cm. below the umbilicus and from 2 to 3 cm. to the right of the midline. The cuticular surface of the skin projects farther toward the center of the circular defect than the deeper layers; local anesthesia.

Division of ileum: After careful evaluation of the circulation of the bowel following this division of the mesentery, a segment is removed between clamps and both remaining ends are closed with Parker-Kerr catgut sutures, each suture being reinforced by three or four Halsted mattress sutures of No. 4-0 Deknatel silk. The distal stump is sutured to the mesentery (Fig. 2) to prevent intussusception, the ties are left long on the upper end to ease re-identification, and both are dropped back into the abdomen.

The resected segment is opened at once by the pathologist and examined for evidences of disease at this level. Presence of diseased bowel at the site of ileostomy favors fecal fistula formation and postoperative diarrhea, and dictates ileac resection upward until good bowel is reached.

Preparation of Circular Ileostomy Incision.—The optimal site for ileostomy on the abdominal wall is dictated in a large measure by the characteristics of the Koenig-Rutzen ileostomy bag. It should be from 6 to 7 cm. below the belt

*The terminal ileum has been involved in one-third of our reported cases.

line, and well away from the anterior superior spine of the ilium and the inguinal fold. In practice the usual position is 6 cm. below the umbilicus and 3 cm. to the right, the same site chosen by Cattell.[4] The skin is infiltrated at this point, picked up, and buttonholed with a scalpel (Fig. 1). A circular defect, two-thirds the original diameter of the ileum to be passed through it, is made in such fashion that the cuticular layer projects farther toward the center of the circle than the deeper layers of the dermis, and may thus later be spread upward along the ileum somewhat (Fig. 1).

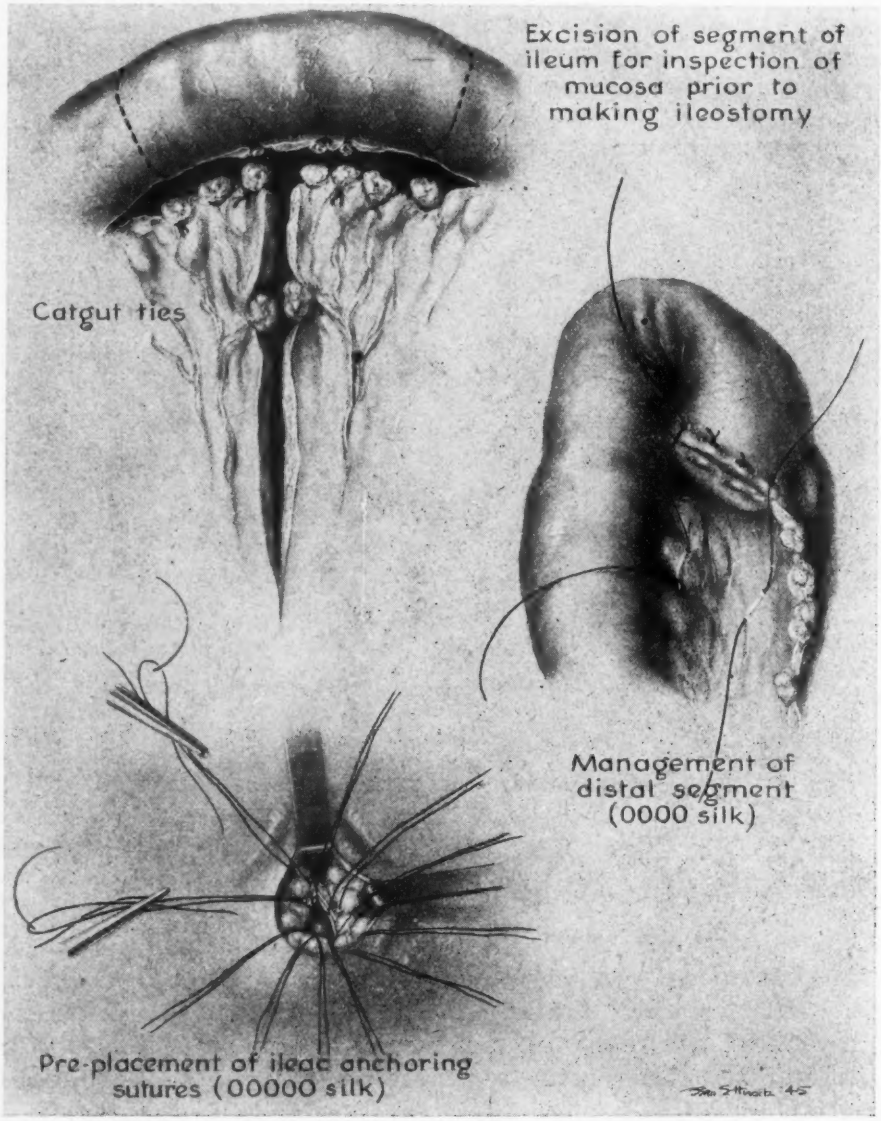

Excision of segment of ileum for inspection of mucosa prior to making ileostomy
Catgut ties
Management of distal segment (0000 silk)
Pre-placement of ileac anchoring sutures (00000 silk)

Fig. 2.—Single-barreled ileostomy. At the site chosen for ileostomy the mesentery is divided 8 to 10 cm. toward the root, using catgut ties. A segment of intestine is removed for immediate gross pathologic examination to rule out ulcerative ileitis at this level. Both remaining ends are closed by Parker-Kerr catgut and Halsted mattress No. 4-0 silk sutures. The distal end is sewed to the mesentery to prevent intussusception. Lower figure shows placement of fine (No. 5-0) silk anchoring sutures in the rectus sheaths and peritoneum.

The rectus sheath is bluntly exposed and longitudinally incised just sufficiently to permit passage through it of a finger of the surgeon equal in diameter to the ileum as first exposed.

Ileac-anchoring sutures: Fixation of the ileum to the abdominal wall by fine silk (No. 5-0 Deknatel) sutures is best accomplished by preplacement of these

sutures, usually from ten to twelve in number, in the rectus sheaths and peritoneum before passage of the bowel through the defect (Fig. 2). Completion of these sutures is accomplished after delivery of the closed ileum by small submucosal bites in the bowel and serosal bites in the mesentery; the bowel is then slipped back into position for tying of the sutures (Fig. 3).

Mesenteric-anchoring sutures: Prior to passage of the ileum through the ileostomy defect, two or three sutures are placed on the mesenteric border and left temporarily without tying (Fig. 3). After securing the ileac-anchoring sutures already described, these mesenteric-anchoring sutures are employed to fix the mesenteric border to the peritoneum and posterior rectus sheath for from 4 to 5 cm. caudad to the ileostomy opening through the abdominal wall in an effort to minimize both prolapse and torsion of bowel about the loop coming to the ileostomy (Fig. 4).

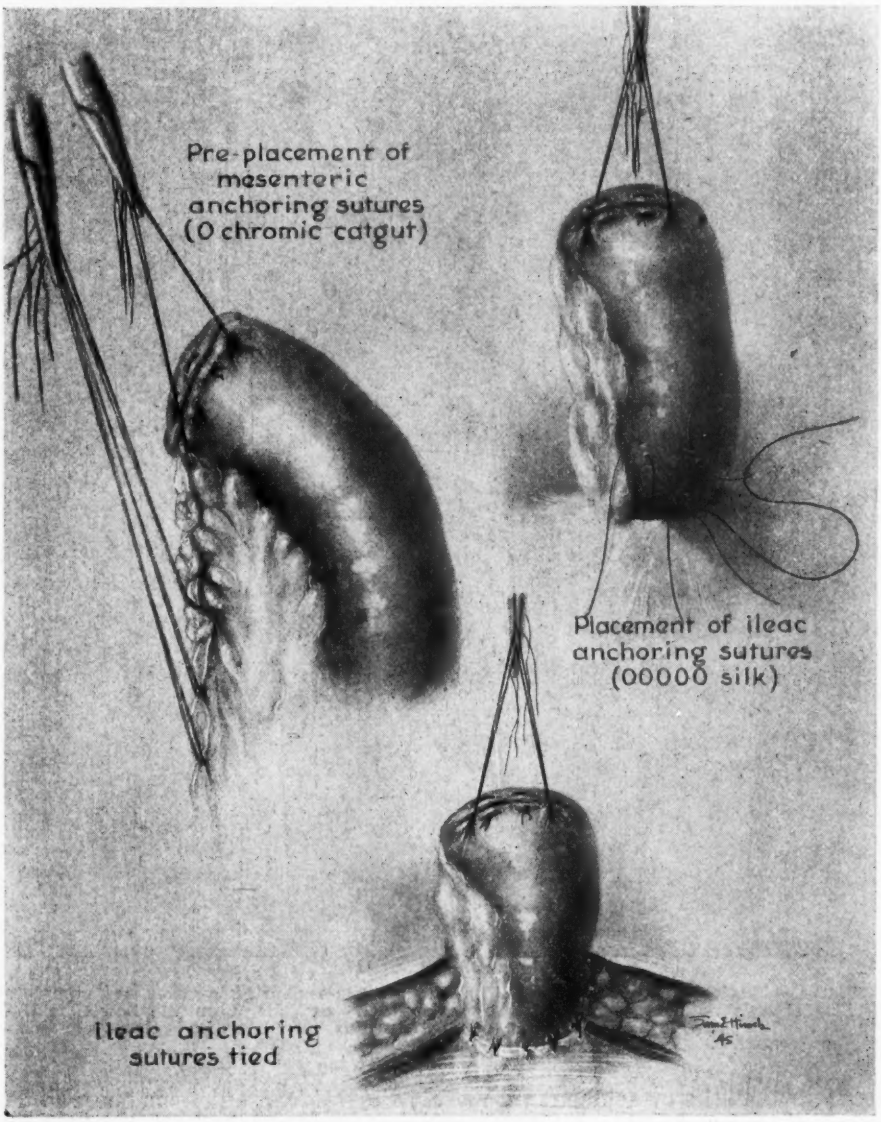


Fig. 3.—Single-barreled ileostomy. Sutures for anchoring the mesenteric cut edge to the anterior abdominal wall are placed before the closed orad stump of ileum is brought through the circular incision, to avoid inaccessible hemorrhage. After passage of the bowel through this incision, the sutures already anchored to the rectus sheath and peritoneum are all completely placed by submucosal bites (*avoiding mucosa*) in the ileum and subserosal bites in the mesentery. The bowel is then slipped back into place, and all sutures are tied and cut.

The ileum should now project about 3 cm. beyond the skin surface, and there should be good color to the bowel and an obvious pulsation of the arteries of the mesentery outside the skin, measures which have been found to militate against later stenosis of the stoma.

Skin sutures: Fixation of the skin edge to the bowel is essential to the subsequent comfort of the patient, as will be indicated. It is best accomplished with No. 000 chromic catgut sutures catching just the cuticular margin. The bowel is worked down into the subcutaneous tissue and the skin edge elevated in the

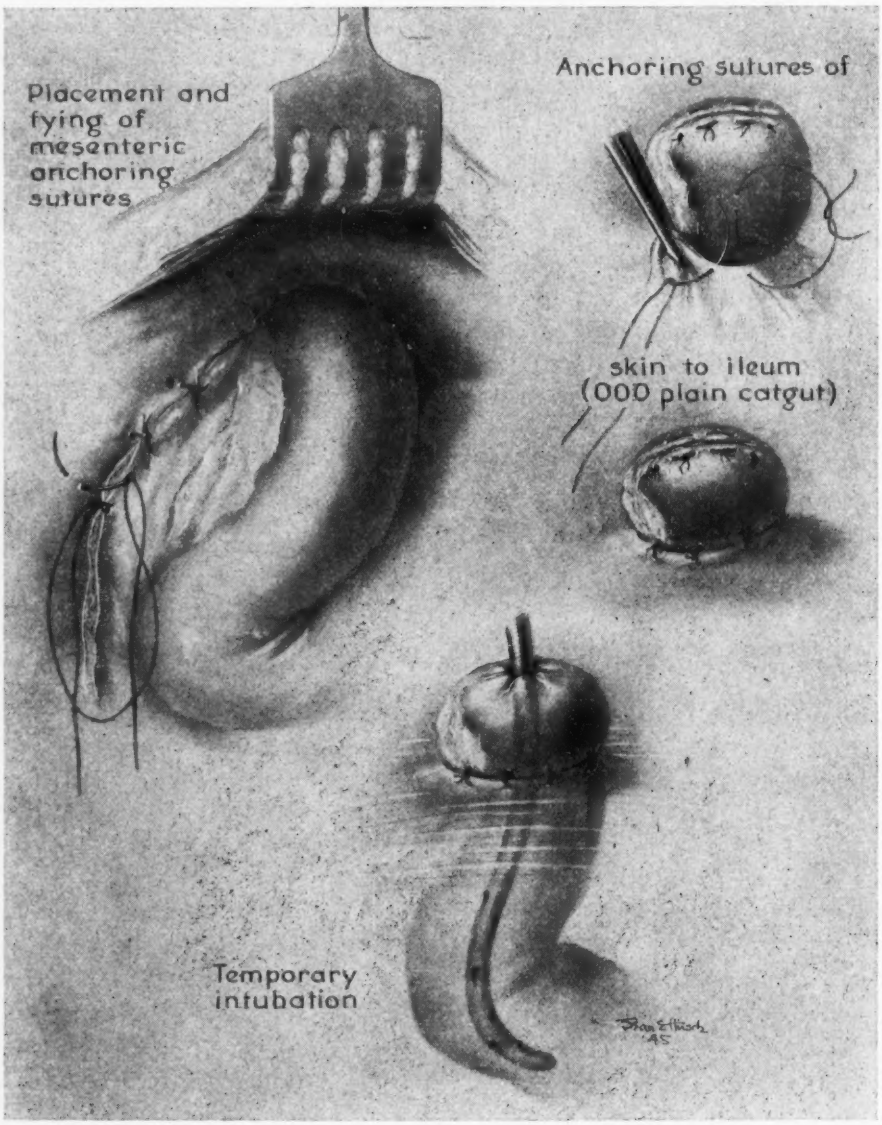

Placement and tying of mesenteric anchoring sutures
Anchoring sutures of skin to ileum (000 plain catgut)
Temporary intubation

Fig. 4.—Single-barreled ileostomy. The mesenteric-anchoring sutures are completed and tied. The skin is sutured to the ileum at a point 1.5 cm. distal on the bowel to the point where it lies naturally. The serocutaneous juncture is covered with petrolatum gauze and the ileum intubated.

process, so that the attachment ultimately lies 1½ cm. out from the point where the skin at first lies naturally against the ileum (Fig. 4). This is best done at once after tying the deep anchoring sutures, before the edema from manipulation is marked. Usually ½ Gm. of sulfanilamide powder is implanted between the bowel and the subcutaneous fat.

The left rectus incision is closed with interrupted silk, No. 000 and 0000 Deknatel, and a dry gauze dressing is applied. Petrolatum gauze is placed around the ileostomy and a No. 14 French, soft rubber catheter with from ten to twelve holes is inserted from 6 to 7 cm. and secured with a catgut purse-string suture (Fig. 4).

Postoperative Care.—

Intubation: The patient returns to the ward awake and comfortable. Full Wangensteen suction is applied to the nasal gastric tube and about one-half suction to the ileostomy catheter. After the first twenty-four hours, appreciable amounts of fluid are usually removed through the catheter. Usually the nasal tube can be removed in forty-eight hours without subsequent discomfort. The ileac catheter must be removed in four days to avoid leakage, and at this time healing is adequate to discontinue aseptic management of both incisions.

Skin care: In removal of the catheter, the following procedure has proved most satisfactory. All dressings are removed, and the skin for from 12 to 14 cm. around the bowel, including the rectus incision, is covered with a ½ cm. layer of the paste described by Ladd and Gross[5] (as used here: aristol 100, castor oil 600, zinc oxide 320, petrolatum 1,280). By the actual cautery the catheter is loosened and the end of the bowel widely opened, care being taken to remove completely the valvelike inverted lips of ileac wall. Drainage is thereafter rather profuse and liquid. The patient now assumes much of the care of the ileostomy, and keeps the skin carefully protected from the ileac secretions by free application of paste and hourly changes of dressings. Nursing help is needed chiefly during the night for the next three days.

About seven days after surgery the patient is fitted with one of a supply of Koenig-Rutzen[6, 7] bags kept on hand for the purpose, and may be up and about the ward (Fig. 5). As soon as the patient masters the management of the bag, usually in another two to three days, he goes home.

Twenty-five days after surgery, shrinkage of the exposed ileum is complete. At this time the patient returns for measurement of the exact outline and position of the mucocutaneous junction, data required for preparation of the patient's Koenig-Rutzen bags.*

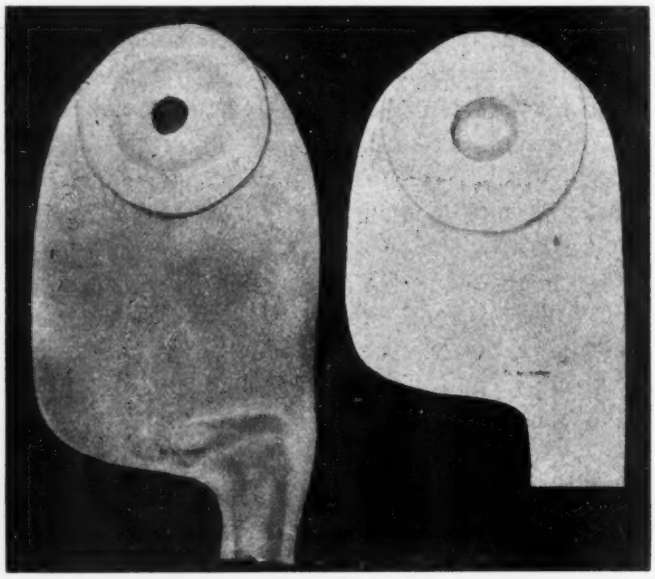
Double-barreled ileostomy: In certain instances stricture or actual obstruction of the colon may be present, or the patient may be too desperately ill to withstand such a procedure as that outlined, even under local anesthesia. In these patients it is safer to perform a double-barreled ileostomy, making the initial 5 to 6 cm. incision under local anesthesia at the site chosen for ileostomy, and filling this incision with the loop of bowel instead of attempting any skin-to-skin approximation at all. A glass rod is passed through the mesentery to support the ileum, but the No. 5-0 silk peritoneal anchoring sutures and catgut skin sutures are used nevertheless (Fig. 6). Spillage into the distal loop can be prevented for about six weeks by a tie of umbilical cord tape around the distal ileum just outside the skin.

Postoperative care is essentially the same as in the single-barreled ileostomies. The bowel is cut down to the glass rod one week after operation before the application of a bag.

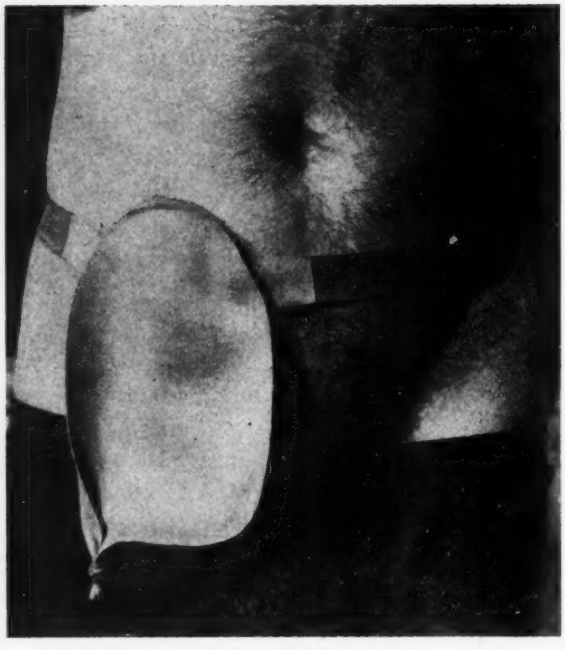
*Obtained from H. W. Rutzen, 1819 Irving Park Road, Chicago, 13, Ill. The patient is supplied with two bags, one for day and one for night use. The bag is made of soft rubber and has a flat brass, rubber-covered facing made to order to fit the patient to hold it against the skin around the stoma. Bland rubber cement is used to fix this facing to the skin for 3 cm. around the mucocutaneous junction. Bags are removed for change each morning and night by application of petroleum naphtha with an eyedropper. They may be emptied between changes through a vent in the lower corner of the bag, otherwise secured by a rubber band.

Rationale of Procedures.—

Anesthesia: Early in the surgical management of the disease in this clinic, the anesthetic of choice was ethylene supplemented with ether. Under this anesthesia one of these debilitated patients died before the skin incision was completed. Another died upon exposure of the peritoneum. More recently we



A.



B.

Fig. 5 *A* and *B*.—The Koenig-Rutzen bag is a rubber-covered brass facing with an opening made individually to fit the bowel, and a bag to hold discharges. It is cemented to the skin, and may be emptied from the lower end when necessary.

have had one apparently good-risk patient go into shock during ileostomy under spinal anesthesia. Another patient went into deep shock following administration of a low spinal anesthetic by a physician-anesthetist, requiring over two liters of plasma and blood to restore the pulse and blood pressure without sur-

gery at all. Two days later, after intravenous infusion of 20 per cent glucose solution, he underwent ileostomy uneventfully under local anesthesia.

Following the adoption of McKittrick's suggestion that ileostomy be done under local anesthesia, no patients have developed shock during this operation. This includes about two dozen cases.

The circular incision: The policy among most surgeons is to perform ileostomy by exteriorization of a loop or end of the bowel through the incision, with closure of the abdominal wall around the ileum. This was the policy at the University of Minnesota Hospitals also, until one unfortunate case of marked wound breakdown led to a review of the cases. The policy since has been to avoid skin-to-skin approximation in the neighborhood of the stoma. Since the adoption of this policy, primary union of both the main and the ileostomy incisions has occurred in every case (Table I).

Double-barreled ileostomy is more commonly used elsewhere than single-barreled ileostomy, but the latter has been preferred here because of greater ease of later colectomy, absolute prevention of passage of ileac contents into the colon, and lower frequency of hernia formation beside the bowel.

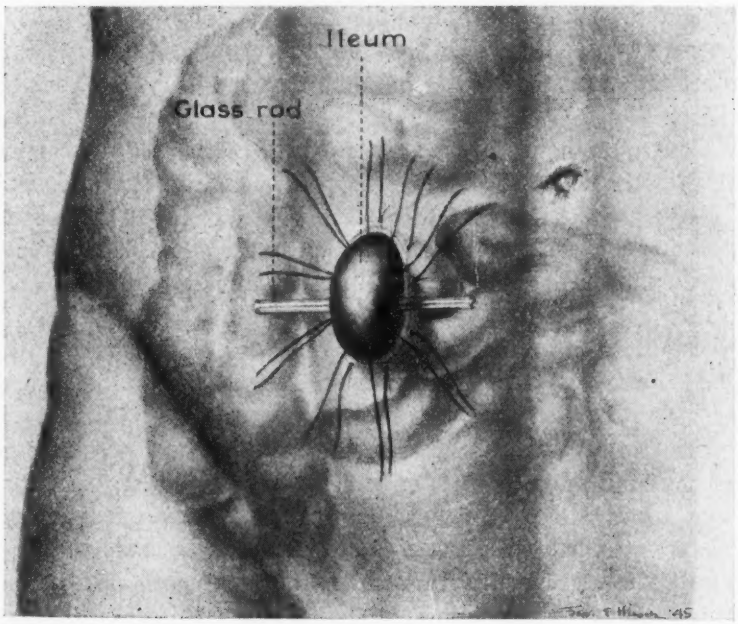


Fig. 6.—Double-barreled ileostomy is reserved for patients with obstruction or too severe a disease to stand the longer procedure. It is performed through a 5 cm. incision which is completely filled by the bowel. Peritoneal and skin sutures are placed as in the single-barreled ileostomy.

Skin care: Skin care according to the measures described has led to virtual freedom from erosions throughout in all but two of the cases in the group of twenty under discussion. In these two there has been temporary erosion, in one case as much as 2 cm. in radius around the stoma, but the erosions have healed well under the regimen outlined.

Several other appliances have been employed. Dressings changed frequently resulted usually in terrific skin excoriation. The Perry cup was demonstrated to us on one patient and fitted on another. It was considered uncomfortable by our patient, and it is less secure than the Koenig-Rutzen bag. The Traveler bag has not been tested here.

Replacement of distal stump inside the abdomen: Lahey[9] and Garlock[10] recommended bringing both ends of the divided ileum out through the skin,

TABLE I. WOUND HEALING IN ILEOSTOMIES FOR CHRONIC ULCERATIVE COLITIS (JAN. 1, 1934, TO MAY 10, 1945)

	TOTAL	PRIMARY HEALING	DISRUPTION OF WOUND	FECAL FISTULA	LATE HERNIA OF PROLAPSE	LATE STENOSIS
Ileostomy made as described	20*	20	0	0	1†	0
Ileostomy made by other methods						
Ileostomy in a long incision	22	14	8‡	0	3	1
Ileostomy through stab wound but sutured from inside abdomen	3	2	1	2	1	0

*Sixteen single barreled; four double barreled. There were two deaths, both in the double-barreled group. One died of a well-organized pulmonary embolus nine days postoperatively. This patient had had strict medical management and absolute bed rest for one month before surgery. The pathologist felt the thrombus had formed before surgery. One comatose late case died five days after emergency ileostomy with purulent ileac drainage and shock. Autopsy showed chronic ulcerative colitis, ileitis, jejunitis, and gastritis.

†Procedure deviated from that described in that the rectus sheath opening was made large enough to admit two fingers.

‡Four wounds healed after one to twelve months. Three operations were redone after six months to three years. One patient died of massive wound breakdown and skin excoriations.

the upper through the incision and the lower through a stab wound elsewhere. This policy has apparently been dictated at the Lahey Clinic by blowout of the closed lower end when dropped into the abdomen. An added stoma to cover and dress seems to us at this clinic to add considerably not only to the difficulty of later colectomy but also to the psychiatric instability of these already insecure patients.

Secure closure methods have been described. Martzloff and Burget[11] found that properly closed loops of ileum, dropped back into the abdomen of dogs, perforated elsewhere than at the suture lines. It has been found in the experimental laboratory here that simple obstruction in the dog's ileum by division and proper closure culminates ordinarily in perforation of the bowel well apart from the suture line.[12]

With these facts in mind, we have had no fear of the procedure described, and have had no reason to regret dropping the closed end back into the abdomen.

Closure of orad stump in preparation of ileostomy: Closure of the orad ileac stump prior to bringing it through the abdominal wall was adopted because it decreased contamination, both from the cut end otherwise held in a clamp of some sort and from tearing of the ileum which was found frequently to occur, in trying to pass it through the necessarily narrow ileostomy opening in the abdominal wall. It also facilitates clean temporary catheter intubation at the end of the procedure.

Fixation sutures: Certain factors have proved particularly important in eliminating prolapse and herniation around ileostomies. The most important of these is the accurate suturing of the wall and mesentery of the ileum to the parietal peritoneum and rectus sheaths just as securely with fine silk sutures as if the bowel were being anastomosed.*

Mesenteric anchorage and prevention of prolapse: Garlock[10] has recommended inclusion of the mesenteric cut edge in the peritoneal closure caudad to the single-barreled stoma as a means of reducing prolapse development. Cattell[4] follows the same procedure. The procedure of suturing this edge to the anterior abdominal wall was adopted in line with this suggestion, and seems to have been partially effective. There has been one case of prolapse for almost

*This policy was adopted after careful dissection of a series of colostomies during the procedure of closure. Certain other observations were made at autopsy and clinically. With but one exception, no colostomy (or ileostomy) was found to be free from herniation beside the bowel unless a colostomy had been performed which included placement of sutures as described. Almost every case of stenosis of colostomies was found to be associated with such herniation and apparently secondary to it. Of those made as described, only one colostomy hernia has developed.

15 cm. in this group, however, and the only factor which seemed likely to play a role in this failure was that the rectus sheath incision had been made large enough to admit two fingers. Since adoption of a rectus sheath opening only large enough to admit one finger equal in diameter to the ileum *as first exposed,* no prolapse has occurred.

Size and type of suture material: Ileostomies were early done with heavy silk sutures. Many of these wounds drained for weeks or months beside the bowel, with discharge of sutures from time to time and great disability, long hospitalization, and psychic disturbance to already unstable patients. Catgut was, therefore, adopted for all sutures and ligatures except the ileac-anchoring sutures, where silk was known to be more reliable. Fine silk sutures of No. 5-0 Deknatel, 1 pound test at this point, are at least as strong as the ileum in which they are placed, and in no instance has use of this size stitch for ileac anchorage been followed by discharge of serum, pus, or suture material from the wound.

Placement of ileac-anchoring sutures: Two ileostomies recently performed by others at the clinic have been followed by development of fecal fistulas, one through the main incision and both through the skin close to the bowel. In one of these the segment employed was involved with extension of the disease from the colon. In both of them the ileac-anchoring sutures were of heavier grade and were placed from within the abdomen, making precise submucosal placement *without entering* the lumen virtually impossible.

In a third case of placement from within the abdomen, the lateral side could not be reached or visualized, and sutures were not placed there. Herniation developed.

The adequacy of the suture method just described is testified by the finding at autopsy of clean healing of the ileum to the abdominal wall in the case of involvement of the entire stomach, ileum, and colon, even though the mucosa was largely ulcerated away and the entire ileac wall heavily involved at the site of ileostomy.

Duration of ileostomy procedure: The rather meticulous procedure outlined cannot be done hurriedly. Under local anesthesia there is little premium on time except for the convenience of the surgeon. The average time including anesthesia in the twenty cases reported in this paper was two hours thirty minutes. Recollection of prolonged suffering from painful open wounds, extensive excoriations, and repeated operations, as well as of the weeks and months I have spent trying to salvage the situation for patients in whom the operative procedures have been done hurriedly and improperly, is adequate incentive to continue to spend this amount of time with them in the proper performance of the ileostomy in the first place.

Skin care: It is true, as much in the postoperative care as in the operative procedure, that a little time spent with the patient is an ounce of prevention.

Various methods of management after ileostomy are feasible. Tincture of benzoin, tannic acid, petrolatum, and vinylite resin[13] have proved entirely inadequate to prevent excoriations. The use of rubber dam cemented to the skin with Koenig-Rutzen cement is satisfactory, but requires close watching and changes at least twice a day. The most useful preparation found so far at this clinic is the paste of Ladd and Gross,[5] which coats the skin with a good protective layer.

It is at this stage that the failure of many ileostomies is caused. *Excoriation and breakdown of the skin are relatively easy to prevent but extremely difficult to heal in the constant bath of irritating ileac discharge.* Early excoria-

tion at this stage or later often may be aided by use of rubber dam precisely fitted around the bowel and cemented to the skin. This may be removed two to three times daily for one-half hour warm baths, with the water level well above the stoma. Dusting the area lightly with zinc stearate powder before application of the skin cement is also helpful. Very occasionally patients have been placed face down on a mattress with a hole in it so secretions fall away without touching the skin.

Once the patient has progressed to use of the Koenig-Rutzen bag, other measures are usually unnecessary. The management is quickly learned by the patient, who is the one person most interested in care of his skin, but in the experience of this clinic is not to be turned over to most internes or to nurses not specially tutored, for trouble and even disaster have almost invariably followed such delegation of care.

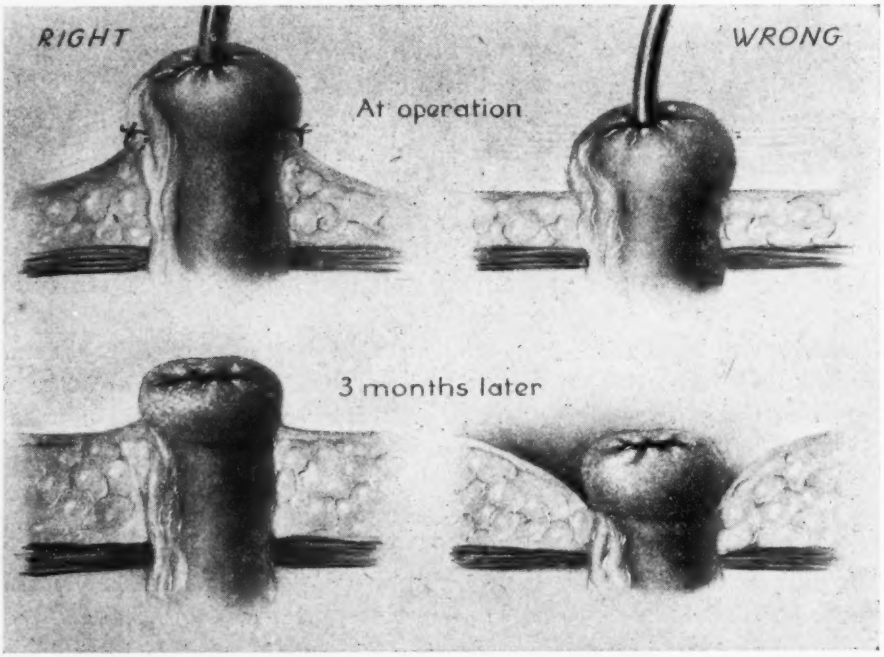


Fig. 7.—At ileostomy the skin is elevated before suture to the bowel. Increase in subcutaneous fat with the usual weight gain then ultimately leaves the patient with the skin flat, so that the bag will fit nicely and excoriation next to the ileum can be avoided. Without this protection, a funnel-shaped depression can defeat efforts to preserve the skin.

Proper choice of time of day, usually three or more hours after eating, so that constant discharge is not occurring, greatly facilitates clean changes of bags.

Thrombophlebitis: Pulmonary embolus has killed two patients here in the last eighteen months, one nine days postoperatively and one preoperatively.* This is such a common complication of the disease that patients are now regularly kept up and active, when possible, up to the time of surgery, and are routinely placed on dicumarol sufficient to keep the prothrombin time twice the control as soon as possible postoperatively.[14]

Postoperative weight gain and skin sutures: When management and care are performed as has been described, most patients have returned to the active

*Embolism also killed the only patient who has been subjected to ileostomy (double barreled) elsewhere by this method. The patient had had a perforation and had a large mass in the sigmoid region. He died following embolism occurring less than seventy-two hours after operation. Dicumarol had been started at sixty hours. It is possible that the drug should be given preoperatively.

life followed before onset of the disease. The weight gain following ileostomy is prompt and marked. Of thirteen patients in whom proper procedures were employed and data are available, the average weight gain in the three months after surgery was 22.7 per cent of the body weight at surgery. The extremes were no gain and 71 per cent gain.

This weight gain is largely due to deposit of fat between the skin and the deep fascia. The result is likely to lead to bulging of the skin except close to the ileostomy, where the surface will necessarily funnel down to the bowel, which, of course, has not increased in length (Fig. 7). In order to permit a flat skin ultimately, to which an ileostomy bag can be well fitted, it is best at the time of ileostomy to lift the skin out immediately adjacent to the bowel and to suture the two thus (Fig. 7).

COLECTOMY

Indications.—Following successful ileostomy, most of these patients not only improve generally, but have a marked diminution in rectal drainage, often to one-half ounce every day or two. This is a reflection of the improvement usually seen in the colon on proctoscopic and on barium enema x-ray examinations. About half of these individuals, however, continue to have enough trouble to indicate colectomy. The indications have been fully discussed elsewhere.[1] In summary they are: persistence of profuse flow, presence of polyps or actual carcinoma, more than one exacerbation of colitis, or profuse bleeding.

General Procedures.—The improvement following ileostomy usually proceeds for three to four months. In order to take full advantage of it, colectomy is deferred until at least five to six months after ileostomy, when it can be undertaken with minimum risk. At this time the patient is usually in excellent general condition and can tolerate general anesthesia nicely.

Initially colectomies were undertaken in stages, such as recommended by Lahey.[9] With the improvement in preparation of patients, the freer use of blood and plasma, and the inception of vastly improved anesthetic measures, the policy has shifted to completion of the job in one stage.

In the earlier cases, the terminal ileum, the colon, and the rectum were removed, the rectum usually in a separate stage. Two difficulties arose with proctectomy. In the first place, a draining sinus was prone to persist for months or years in the perineum. In the second place, those males subjected to the operation were found to be incapable of penile erection. Further experience suggests that the rectal pouch can be easily watched from time to time by proctoscope and that it heals fairly often sufficiently to permit consideration of anastomosis to the ileum.

Experience is accumulating to show that it is not possible to determine by barium enema-roentgen study in chronic nonspecific ulcerative colitis if some portions of the colon are normal. Repeatedly, efforts have been made to save portions of the colon on this basis. Except in the rare segmental colitis, no cases have been found in which portions of the colon could be salvaged when x-ray alone indicated them normal. In the rectum and lower sigmoid, proctoscopic examination is reliable. The colon is now removed to the rectosigmoid, therefore, and the rectum observed periodically by proctoscope.

The procedure has been reduced routinely to excision of terminal ileum and colon to the pelvic floor sparing the rectum. With such measures, closure of the stump by Parker-Kerr sutures or the Petz sewing machine and silk mattress sutures was difficult and unsatisfactory. The wall of the rectosigmoid is usually heavily inflamed, edematous, thickened, and friable, rendering inversion virtually impossible by standard methods. Two out of four of the rectums closed

by those methods, before adoption of the present technique, developed pelvic abscesses requiring drainage through the rectum or vagina.

Rectosigmoid Closure.—Because of these difficulties, a closure was devised which would eliminate difficulty with inversion and eliminate the dead space usually remaining between the first and second rows of sutures.

The colon is removed to the apex of the sigmoid in customary fashion, making no effort to peritonealize the lateral gutters. Leaves of peritoneum are elevated from the right and left sides of the mesocolon down to the pelvic floor for subsequent reperitonealization, but the superior hemorrhoidal vascular supply to the rectosigmoid is carefully preserved (Fig. 8). Two heavy ties (No. 9 silk) are placed 1½ cm. apart around the carefully devascularized bowel at about the second sacral level, and the gut is cut between them with the cautery.

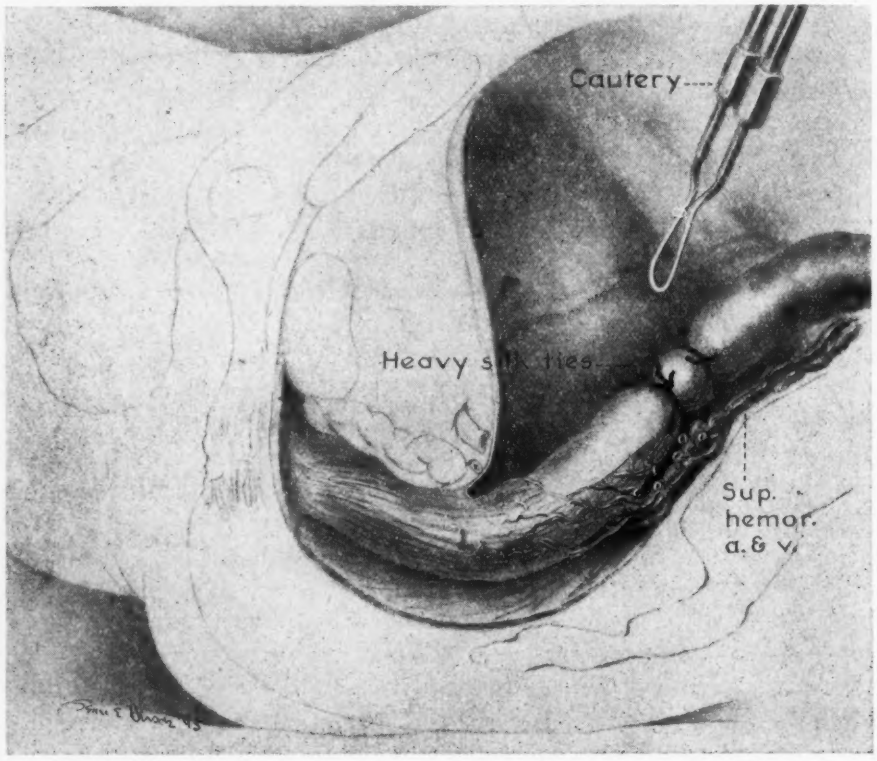


Fig. 8.—Closure of thickened rectal stump in ulcerative colitis. Superior hemorrhoidal blood supply is preserved. Two heavy ties are placed around the carefully cleaned rectosigmoid, 1.5 cm. apart. The bowel is cut between with the cautery.

An assistant now inserts into the rectum from outside the sterile field a brass tube 1 cm. in diameter fitted with a loosely fitting 7 mm. wooden core. This procedure is facilitated by elevation of the hips preoperatively on several folded sheets. This instrument is inserted until the tip can be palpated by the operator from within the abdomen.

Two straight needles are threaded, one on each end of a one-meter piece of No. 30 stainless steel wire. One is inserted through the cut end of the colon and forced well into the wooden core; it passes through the loop of heavy silk which encircles the gut, close to it behind. The second needle passes through the bowel wall anteriorly, but just outside the silk tie, and passes into the core (Fig. 9).

The brass tube is now held in place while the wooden core is withdrawn with the needles. The assistant outside the sterile field can now put tension on the

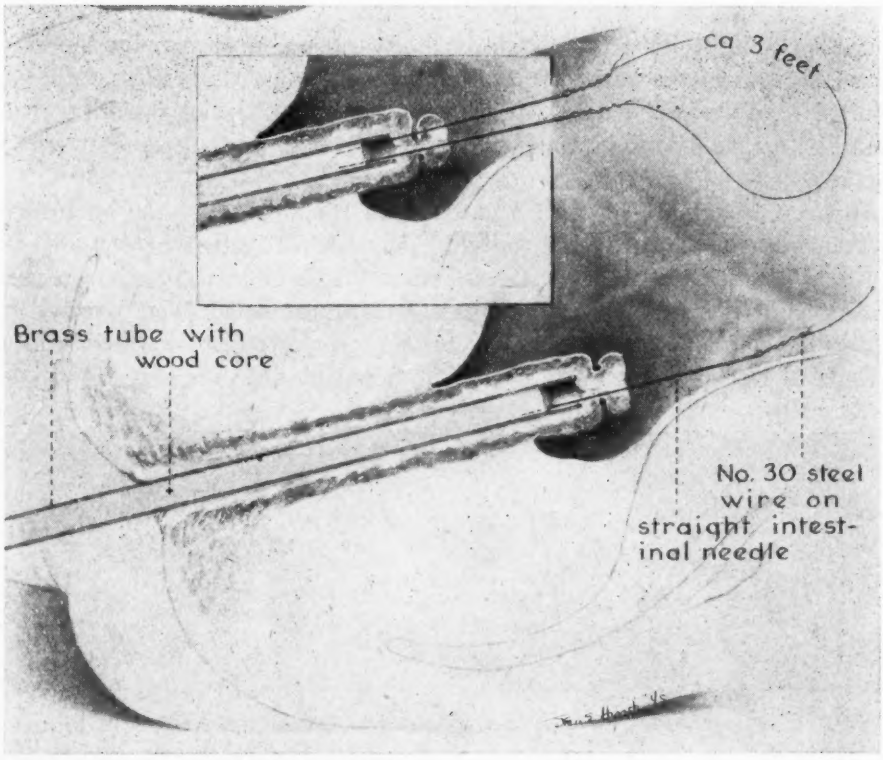


Fig. 9.—Rectal closure in ulcerative colitis. A metal tube with a loose wooden core is inserted from outside the sterile field into the rectum. A 3 foot piece of No. 30 steel wire is fitted with a straight needle on each end. These are passed through the bowel and forced into the wooden core in such a fashion that, when the core is withdrawn, the wire will pull on as much tissue as possible and pass out and through the heavy silk tie.

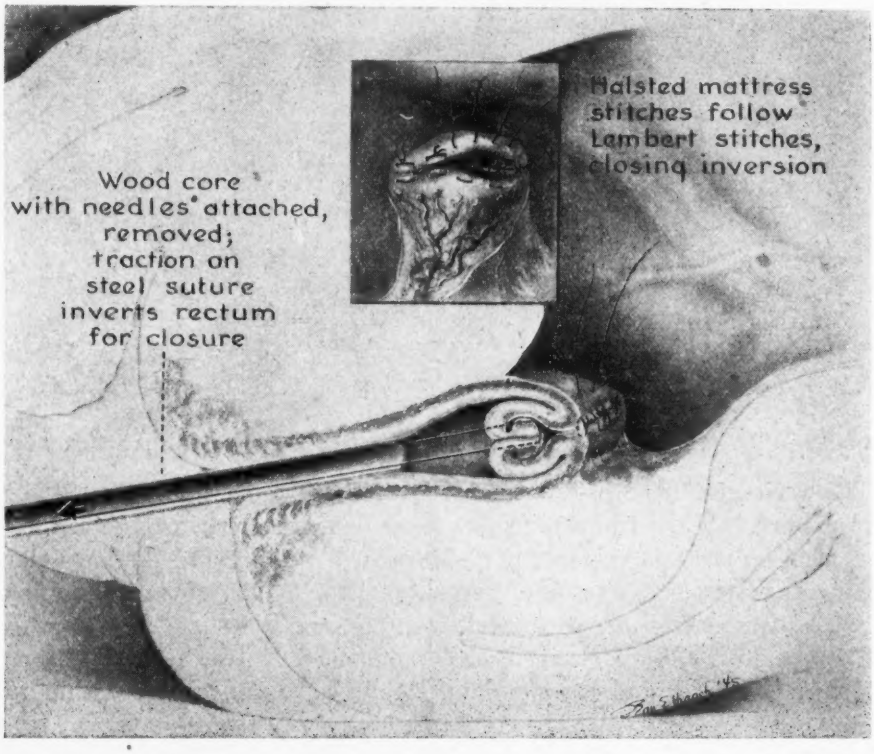

Halsted mattress stitches follow Lambert stitches, closing inversion
Wood core with needles attached, removed; traction on steel suture inverts rectum for closure

Fig. 10.—Rectal closure in ulcerative colitis. By traction on the wire from below, inversion of the thickened narrow rectum is possible. It is secured by a row of Lembert and a row of Halsted mattress sutures.

ends of the steel wire to accomplish, by use of some force, inversion which would otherwise be impossible. Such tension accomplishes inversion by the employment of sutures in the bowel wall for countertraction. Closure is completed by a row of Lembert silk sutures and another of Halsted mattress silk (No. 4-0 Deknatel). Usually 50 mg. of sulfathiazole are implanted between rows (Fig. 10).

In order to open the contaminated dead space between the encircling tie and the two-layer closure, the encircling silk and much of the enclosed bowel are cut by a sawing motion (that is, similar to use of a Gigli saw) on the two ends of the steel strand by the assistant outside the field. The inverted bowel can now be palpated through the rectal wall to have been turned inside out. No dead space remains, and healing should be uncomplicated (Fig. 11).

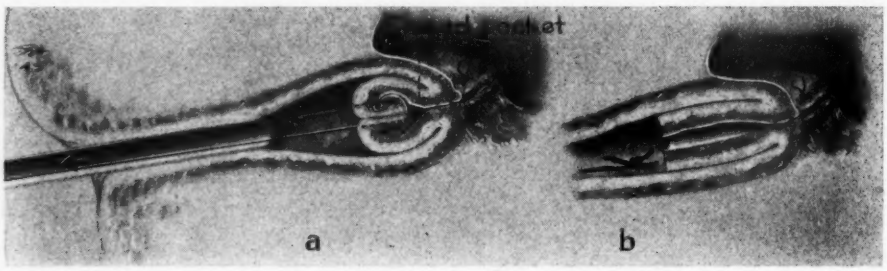


Fig. 11.—Rectal closure in ulcerative colitis. Tension on the rectal wall by the mass of inverted tissue, and the closed contaminated space between the encircling tie and the two-layer closure, are both eliminated by cutting the encircling tie and completely inverting the cut end by means of the steel wire.

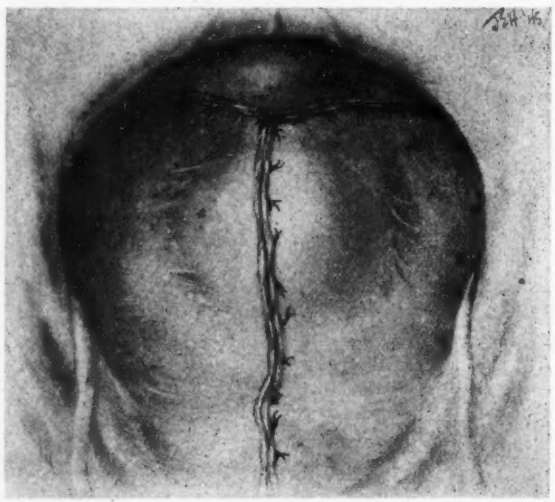


Fig. 12.—Pelvic floor is reconstituted, using interrupted Halsted mattress silk sutures.

The peritoneal floor is refashioned with interrupted Halsted mattress silk sutures, which hold even in case of pus formation, using peritoneum of the bladder in men, or posterior surface of the uterus in women, and the flaps elevated from the mesosigmoid (Fig. 12). Usually about 2 Gm. of sulfathiazole have been implanted beneath the peritoneal closure.

To date, five closures have been accomplished in this fashion. None has had pelvic infection, and the patients usually have gone home within twelve days.*

*Recently a modification of this method has been tested to open the double diaphragm in utilization of the Halsted bulkhead anastomosis[15] to reestablish continuity after resection of a low-lying rectosigmoid carcinoma. The wire kinked and broke, so that the diaphragm had to be punctured with a knife, but a clean anastomosis and primary union resulted.

Risk of Colectomy in Chronic Ulcerative Colitis.—The risk of colectomy in ulcerative colitis is variously estimated.[16] Apparently it is no greater than that of laparotomy. There have been no deaths in this group at the University of Minnesota Hospitals since Jan. 1, 1934. All patients colectomized here for ulcerative colitis in this period are alive at the present time.

RE-ESTABLISHMENT OF CONTINUITY

To date, six patients with ileostomy for chronic ulcerative colitis have had restoration of continuity performed by ileoproctostomy. Four of these were done by Owen H. Wangensteen and two by the author. In general, when the procedure has been reserved for those who develop solid stools from the ileac stoma and who present rectal mucosa normal to proctoscopic study, the patients have remained well without diarrhea. Those who do not satisfy these criteria appear to redevelop diarrhea and ulcerative lesions in the rectum, although the mucosal area involved is too small to interfere appreciably with the patient's general health.

We have had no experience with simple closure of ileostomy at this clinic.

CONCLUSIONS

1. With prompt and proper surgical management, chronic ulcerative colitis is not a disabling disease.

2. Ileostomy is the first surgical procedure in chronic ulcerative colitis almost invariably at the University of Minnesota Hospitals and should not be complicated by any other simultaneous procedures.

3. A method of ileostomy which has proved successful in this reported series of cases is presented. In twenty cases there were one case of prolapse, two cases of recession, two cases of some trouble with skin excoriation, no cases of failure of primary union, no hernias, and two deaths. The deaths occurred from pulmonary embolus in one case and from involvement of ileum, jejunum, and stomach in the other.

4. In the experience at the University of Minnesota Hospitals the Koenig-Rutzen bag offers the only satisfactory method of handling properly constructed ileostomies.

5. Dicumarol postoperatively is given routinely to patients with chronic ulcerative colitis.*

6. Colectomy is done four to six months after ileostomy if there are: repeated exacerbations of colitis, polyps or carcinoma of the colon, profuse drainage, or continued bleeding. The rectum is spared and watched.

7. A method of closing the rectal stump is presented. It appears to be safer than older methods.

8. In selected cases, re-establishment of continuity has been possible by ileoproctostomy and resection of the intervening bowel many months after ileostomy.

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*Recently it has been given preoperatively as well.

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EXTRUSION OF GASTRIC MUCOSA THROUGH THE PYLORUS

REPORT ON TWO PATIENTS TREATED BY PARTIAL GASTRECTOMY

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THE basis for gastrointestinal symptoms is frequently difficult to determine. This is because there is a multiplicity of factors playing a role in the production of symptoms. These factors fall into two groups: those that have a definite demonstrable physical basis and those in which no physical factor is found. Before a patient is placed in the second category, it is important that every diagnostic aid be employed to demonstrate that there is no physical basis for the patient's complaints. At the United States Naval Hospital in Seattle a few patients who had been labeled malingerers were found to have extrusion of gastric mucosa into the pylorus, which was the cause of their symptoms. Since operation these men have been restored to duty.[10]

This presentation is concerned with a small group of patients having gastrointestinal disorders which are difficult to diagnose, namely extrusion or prolapse of gastric mucosa into the pylorus causing obstruction. Two patients treated by partial gastrectomy are reported.

According to Meyer and Singer,[1] the first instance of extrusion of gastric mucosa was reported by Von Schmieden[2] in 1911. Since then the following reports have been made: Eliason and Wright,[3] 1925, one; Eliason, Pendergrass, and Wright,[4] 1926, two; Meyer and Singer,[1] 1931, one; Meyer,[5] 1935, one; Pendergrass and Andrews,[6] 1935, three; Rees,[7] 1937, four, three operated upon; Bohrer and Copleman,[8] 1938, one, died of complications; Archer and Cooper,[9] 1939, four. We are reporting two and Commander A. M. French, United States Naval Hospital, Seattle, has described four, as yet not reported. These make a total of twenty-three proved by operation.

Symptoms of extrusion of gastric mucosa into the pylorus are indefinite. This is because it depends upon the degree of obstruction present. Complaints may be classified under four headings.[7] (1) Periodic upper abdominal distress,

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with sensation of fullness in this region; (2) cramplike pains after eating, more pronounced after taking solid foods; (3) vomiting, the last symptom to appear; and (4) loss of weight, the logical result of the first three symptoms. These patients are usually afraid to eat, at first because of pain and later due to vomiting. As the symptoms are intermittent, patients have varying periods in which they feel well; however, the tendency is to become worse.

Radiologic findings are somewhat indefinite.[3, 4, 6, 7, 11] In a typical case there is a shadow containing a small, opaque streak in the duodenum. This streak differentiates it from tumor. It should be emphasized that the findings under fluoroscopy may be inconstant during any given examination. Radiologic examination is the only method by which diagnosis can be made. The radiologist is likely to miss the diagnosis unless he is aware that such a condition exists and looks for it.

ETIOLOGY

Eliason and Wright[3] believe that this condition is the result of chronic inflammation causing hypertrophy of gastric mucosa with extrusion of the mucosa into the pylorus. Rees[7] is of the opinion that the pathology is due to narrowing of the pyloric outlet following hyperperistalsis in an effort to push the stomach contents through. The condition must not be confused with Billroth's hypertrophy, which is a benign hypertrophic pyloritis. According to Pendergrass,[12] and Archer and Cooper,[9] bleeding is occasionally associated with this condition, causing secondary anemia. They state that hypertrophy of the gastric mucosa may undergo malignant change.

REPORT OF CASES

CASE 1 (Hospital No. 446341).—Mrs. S. M., a 61-year-old woman presented herself to one of us (I. S.), May 22, 1944, complaining of pain in the epigastrium for the past week. The pain was intermittent and taking food made it worse. It made no difference what kind of food she ate. She had vomited several times.

Past history brought out the following: twenty-five years before she had had cholecystostomy; eight years previously, cholecystectomy; five years before, hysterectomy was performed for bleeding fibroids. The maximum weight had been 204 pounds two years previously. At that time she had attended the diabetic clinic at the King County Hospital. The diabetes mellitus was of a mild degree, and insulin did not have to be administered. The diabetic condition had been under good control until the present symptoms developed.

Physical examination revealed a woman weighing 163 pounds. The skin was clear and there was no evidence of icterus. The blood pressure measured 136/90 mm. of mercury. General examination brought out the following: there was nothing abnormal about the head, neck, or chest; the heart was apparently not enlarged; there were two scars on the abdomen, an upper right rectus incision, and a lower midline incision extending from the umbilicus to the symphysis pubis. Both were well healed and showed no evidence of herniation. There was no evidence of fluid or tumor in the abdomen. Pain in the epigastrium was elicited upon deep palpation. Pelvic examination revealed the uterus to be absent, the cervix present, but with no evidence of pathology. Rectal examination was negative; the extremities were unchanged and the reflexes normal.

Laboratory Examination.—The blood Wassermann was negative. There was no sugar in the urine. Fasting blood sugar revealed the blood level to be 120 mg. per hundred cubic centimeters of blood. The results of gastric analysis were 75 free HCl, total 90. Radiologic examination at this time revealed an "area of roughness in the pyloric end of the stomach." This radiologist concluded that while he could not demonstrate ulcer, it was the most likely diagnosis. Flat plate of the abdomen revealed no evidence of stones in the kidney, ureter, or common duct.

The patient was placed under treatment for ulcer without relief. Codeine sulfate had to be taken frequently to control pain. At times she felt well, and again, especially upon taking solid foods, she had severe pain. However, her appetite was good, but she was afraid to eat. She continued to lose weight.

Oct. 6, 1944, she was referred to another radiologist, who reported that the esophagus showed no abnormality. The one abnormality, as seen at time of fluoroscopy, was extrusion of redundant mucosa in the distal end of the stomach through the pylorus into the base of the cap. The small intestine distal to the duodenum showed no abnormality. The examination was repeated by the same radiologist with the same findings. Examination of the small and large intestines revealed no abnormality.

The symptoms of pain, nausea, and vomiting continued intermittently. Nov. 11, 1944, she was examined by one of us (M. N.) before entering the hospital for operation. Her weight was 134 pounds. At that time no additional physical findings were revealed. Nov. 25, 1944, under combined spinal pontocaine and intravenous sodium pentothal anesthesia, exploration brought out the following: the gall bladder was absent; there were adhesions in this region; the common duct was not dilated. Careful palpation revealed no evidence of stone or obstruction. Aspiration of the common duct revealed normal bile. The liver was not enlarged and showed no gross pathologic change. The uterus was absent. Nothing abnormal was found in the small or large intestine. The spleen was not enlarged. When the stomach was examined it was found to contain a soft, tumorlike mass which could be pushed into the pylorus; otherwise, the viscus was negative.

What Was Done.—Three-fifths of the distal part of the stomach were resected. A retrocolic anastomosis was done according to the Finsterer method. Five grams of sulfathiazole crystals were sprinkled in the upper abdominal cavity and the abdomen was closed without drains. Pathologic examination brought out the following: before fixation of the gross specimen, redundance of gastric mucosa and almost complete obstruction of the pylorus could be demonstrated. During microscopic examination, section of the pyloric end of the stomach showed the mucosa with a moderate amount of monocytic infiltration between the gland structures. The musculature was normal. Diagnosis was chronic gastritis.

Postoperative convalescence was uneventful. She was discharged to her home on the eleventh postoperative day. On the fifteenth postoperative day she developed a slight icterus which disappeared in five days. This was thought to be due to sulfathiazole. She remained well and was completely relieved of the symptoms until Dec. 28, 1944, when she developed pyelonephritis and diabetes mellitus, and was readmitted to the hospital. The kidney infection was difficult to combat, but she gradually improved and was discharged from the hospital Jan. 30, 1945. She remained well and was relieved of all preoperative symptoms until March 26, 1945, when she developed epigastric pain, nausea, vomiting, and prostration. She was taken to the King County Hospital where, in spite of administration of glucose, plasma, and transfusions, she became progressively worse. The blood protein remained low despite these measures. Generalized edema developed. She was unconscious for days at a time. Clinical jaundice was present throughout her stay in the hospital until she died, April 29, 1945. Postmortem examination revealed subacute hepatitis, fibrosis of the pancreas, terminal bronchopneumonia, general anasarca, and large decubitus ulcers. The stomach showed no change except subtotal resection. There was no evidence of ulcer or obstruction.

CASE 2 (Hospital No. 45948).—J. V., a 42-year-old fisherman, was first seen May 7, 1943, at which time he complained of epigastric pain and nausea of one week's duration. He had had no previous stomach trouble, no melena or change in bowel habits. Past history revealed that in 1928 he had had an appendectomy and at the age of 15 a mastoidectomy of the right side.

Physical examination at that time revealed a man weighing 210 pounds, which was his usual weight, and measuring six feet in height. There was a mastoidectomy scar on the right side; otherwise, there was nothing abnormal about the head, neck, or chest; the blood pressure measured 124/86 mm. of mercury; pulse was 70; heart was not enlarged; there was a scar on the right side of the abdomen which was healed well; there was slight tenderness to deep palpation in the epigastrium. No fluid or tumor masses were felt in the abdomen. There were no hernias. Genitals were normal. The extremities were unchanged. The reflexes were normal.

Laboratory Examination.—The blood Wassermann was negative; blood count and urinalysis were within normal limits; gastric analysis free HCl 60, total 85. Radiologic examination, May 8, 1943, revealed a small penetrating ulcer on the lesser curvature of the stomach at the angle. The duodenal cap was normal. The small intestine distal to the duodenum was normal throughout.

The patient was placed under treatment for ulcer by one of us (I. S.), including alkalies and antispasmodics. Radiologic examination, June 19, 1943, by the same radiologist revealed no evidence of ulcer.

The patient was next seen Feb. 12, 1945, at which time he complained of pain, nausea, and vomiting, especially after eating solid foods. Because of this condition he had become afraid to eat. While he was relieved by food at the time he had gastric ulcer in 1943, food now definitely made him worse. He had lost twenty-four pounds within a period of six weeks, which he stated was due mainly to the fact that he was afraid to eat. Physical examination brought out the following: weight, 186 pounds; there was nothing abnormal about the head, neck, chest, or abdomen except that there was slight tenderness to deep palpation in the epigastrium. The blood pressure measured 120/84 mm. of mercury. The temperature was normal; blood count and urinalysis were within normal limits. Radiologic examination at this time revealed: The barium passed readily through the esophagus, which appeared normal. The stomach showed definite prepyloric induration strongly suggestive of early carcinoma. There was marked concavity of the base of the bulb similar to the appearance produced by prolapse of the gastric mucosa, and it was possible that all the findings were due to such a condition. Carcinoma seemed more probable, however. At the end of three hours the stomach was almost empty and the head of the meal was in the cecum.

The patient was operated upon Feb. 23, 1945, by one of us (M. N.), at which time the following was found: in the prepyloric region there was a tumorlike mass which was firm and which could be pushed into the pylorus. At the angle there was a small scar, probably representing a healed gastric ulcer. Further exploration of the abdomen revealed no evidence of gall bladder or biliary tract disease, and there was nothing abnormal found in the small or large bowel.

What Was Done.—Under combined spinal pontocaine and intravenous sodium pentothal anesthesia, an upper left paramedian incision was made. Approximately three-fifths of the distal end of the stomach were resected and an anticolic anastomosis was done. The abdomen was closed without drains. Examination of the removed segment of the stomach before fixation revealed marked hypertrophy of the prepyloric gastric mucosa, and this could be easily pushed into the pylorus causing complete obstruction. There was no evidence of ulcer of the stomach. During histologic examination, section made from the stomach wall near the pylorus showed a large amount of monocytic infiltration, germinal center formation, and destruction of much of the mucosa due to the infiltration of monocytic cells. The whole thing indicated a marked gastritis at this end of the stomach. Diagnosis was gastritis.

The patient made a good recovery and was discharged to his home on the tenth postoperative day. Since that time he has been completely relieved of the preoperative symptoms of pain, nausea, and vomiting. He has gained ten pounds in weight and feels well.

DISCUSSION

At present the surgical treatment recommended for this condition is resection of the redundant gastric mucosa.[7-9] This is done by making an incision through the anterior wall of the stomach and placing a finger into the pylorus through this opening. A second incision is then made into the pylorus. The redundant gastric mucosa is excised and the incision in the mucosa is sutured. All bleeding vessels are ligated and the incision in the pylorus is closed. Some surgeons apparently close this in a longitudinal manner and some transversely. Finally the incision in the stomach is closed.

Rees[7] reports a patient on whom he performed posterior gastrojejunostomy with no relief. This is what one would expect, as the peristaltic action of the stomach would still push the redundant mucosa into the pylorus. Three years later resection of gastric mucosa was done without relief. This is evidently the only patient reported who did not obtain relief from resection of gastric mucosa. Rees states that ". . . the closure of the gastric mucosa did not hold entirely," as revealed by subsequent x-ray findings, and attributes failure to this cause.

Gastric resection was done in our first case because of the high acidity. It was feared that ulcer might develop at the side of the excision of the gastric mucosa or that closure of mucosa would not hold. It was also deemed advisable to perform an operation that would give the patient the best possible chance for permanent relief. In Case 2, gastric resection was done in view of the history of gastric ulcer, high acidity, and the radiologist's report of possible car-

cinoma. Besides, subtotal gastrectomy for benign lesions is now being done with a great deal of safety so that the added risk of gastrectomy over excision of the mucosa is minimal. In young patients with moderate acidity, excision of mucosa is no doubt the operation of choice.

It is our opinion that redundance of gastric mucosa is not as uncommon as reports would indicate. Some patients who have mild gastric symptoms where nothing definite is found may be suffering from this condition. Every abdominal surgeon has no doubt noted redundance of prepyloric gastric mucosa when palpating normal stomachs. Therefore, medical management of these cases should be instituted, especially if the symptoms are not severe. This should be done even if the actual condition has been demonstrated radiologically. In this connection, Bockus[13] in his book, *Gastro-Enterology,* stated, "Moderate redundancy of the pyloric mucosa is probably not uncommon but, unless it is associated with severe gastritis or actual prolapse of the gastric mucosa into the duodenum in sufficient quantity to interfere with evacuation of the stomach contents, symptoms are not produced."

The pathologic study of the tissues removed in our cases would indicate that the condition is probably due to inflammation. There was nothing to indicate a narrowing due to hypertrophy or contraction of the musculature in the pyloric region. In Case 2, especially, the inflammatory reaction was marked.

Although we are not responsible for the radiologic diagnoses in these cases, attention should be called to the fact that many radiologists are not aware of the existence of this condition and therefore are not on the lookout for it. The first radiologist who examined the patient in Case 1 was apparently unable to interpret what he saw. Consequently the diagnosis was missed, although the condition was probably observed by him. On this subject Bockus[13] stated, "When complete prolapse into the duodenum occurs, the differentiation between pedunculated tumor and prolapsing gastric mucosa will often be impossible and operation may be necessary. If there is a large defect in the pyloric canal and a negative shadow in the duodenal bulb, the condition may not be distinguished from that of pyloric ulcer, hypertrophic gastritis, or carcinoma."

It will be noted that we have used "extrusion" instead of "prolapse" to designate the condition present in these cases. The term "prolapse" is defined in the dictionary[14] as: "The falling down or sinking of a part or viscus." Therefore, it does not describe this condition accurately. "Extrusion," as defined by Dorland[14] is, "A pushing out; a forcing out or expulsion"; consequently it conveys correctly the pathologic physiology present in these cases, as it is due to the peristaltic action of the stomach pushing the hypertrophic redundant gastric mucosa into the pylorus that causes obstruction and symptoms.

SUMMARY AND CONCLUSION

1. Two cases of extrusion of gastric mucosa into the pylorus causing intermittent obstruction with marked loss of weight are reported. Partial gastric resection was done in both cases with satisfactory results, although one patient died five months after surgery from hepatitis.

2. The condition is undoubtedly more prevalent than reports would indicate. Diagnosis is made by the roentgenologist who must be aware of and look for this condition.

3. In mild cases, medical management should be instituted before surgery is recommended. Two operative procedures are at the surgeon's command: re-

section of redundant gastric mucosa or partial gastric resection. The choice of the procedure should be determined by the age of the patient, the gastric acidity, and the judgment and skill of the surgeon.

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INTUSSUSCEPTION IN ADULTS

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STATISTICAL data appear to show that of all intussusceptions the incidence in adults is about 5 per cent.

In infants, according to Perrin and Lindsay, 78.5 per cent occur in the sixth and seventh months of the first year, especially in boys. The ileocecal and ileocolic varieties are the most common at all ages, but enteric intussusception occurs more frequently in adults than in children.

Incidence.—A comparison of the incidence of intussusception according to Perrin and Lindsay and Fitzwilliams is shown in Table I.

It is common medical experience that there are, as a rule, no discoverable etiologic factors in infants, while in adults there is usually an organic lesion, a benign or malignant tumor, as the basic cause.

Other provocative influences are (1) typhoid ulcers, (2) tuberculous ulcers, (3) dysentery, (4) acute appendicitis, (5) Meckel's diverticulum, (6) a con-

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TABLE I. INCIDENCE.

	PERRIN AND LINDSAY (4,000 CASES) (PER CENT)	FITZWILLIAMS (1,000 CASES) (PER CENT)
Ileocecal	46.5	60.8
Ileocolic	37.6	25.6
Enteric	10.1	0.0
Colic	5.6	7.3

genital ileal band, (7) a submucous lipoma of the ileum, (8) a foreign body, (9) a sudden radical dietary change (Moore), or (10) neoplasms.

Enlarged lymphoid patches not infrequently cause intussusception, and an instance in point appears to be the high incidence of the ileocecal variety, with the presence of large Peyer's patches. The detrusor action of the terminal ileum and the greater size of the colon have been regarded as other probable causes. Lymph patches or polyps, it is maintained, irritate the intestine and thus provoke peristalsis. Wardill[1] suggested "fixed spasm" as the prime mover.

"Primary" intussusception does not commonly occur in the adult in contradistinction to the condition in infancy because lymphoid tissue becomes progressively reduced with age and also because the lumen of the intestine in the adult is relatively larger.

The majority of intussusceptions are of the enterocolic variety, and it is in the ileocecal zone that considerable anatomic alteration in the size of the gut takes place.

The Anatomic Causation.—It is a widely accepted view that an intussusception is induced by irregular and increased intestinal peristalsis due to some irritant or lesion in the wall or lumen of the intestine. The condition in adults is rarely functional.

Physiologic Causation.—Fraser[2] proved that the innervation of the ileocecal zone is distinctive in that the nerve supply of the ileum is sympathetic and parasympathetic; the cecum receives a supply of sympathetic nerves only. An explanation is thus found for the descending peristaltic irregularity.

Pathologic Causation.—Intussusception at times occurs during an attack of enteritis and there is a strong probability that the swollen Peyer's patches may induce the hyperperistalsis. (It has been shown frequently that there is a distinct concurrence of ileocecal tuberculous lesions and intussusception.)

Intussusception of the gastrointestinal tract may take place anywhere between the stomach and the rectum. The large bowel is the most frequent site of involvement in adults because benign and malignant growths, particularly the former, are more common there than in any other part of the gastrointestinal tract.

An intussusception usually forms a rather firm sausage-shaped swelling composed of three concentrically arranged tubes or layers—the afferent, efferent, and ensheathing. The first two form the intussusceptum and meet at the apex, or most distal part of the invagination; the ensheathing layer constitutes the intussuscipiens and joins the returning layer at the neck of the intussusception. With the growth, in length, of the invagination, the mesentery is progressively drawn into the intussusceptum where it is caught between the entering and returning layers on the concave side.

The constriction and torsion of the mesentery induce congestion from pressure on the veins, which is most obvious at the apex and in the adjacent part

of the returning layer. The apical part particularly swells to a great extent. This is due to the extravasation of blood and mucus obstructing the lumen. Adhesions subsequently form between the apposed serous surfaces of the entering and returning layers.

Varieties of Intussusceptions.—Intussusception may be single or compound. In the latter, the primary invagination is surrounded by a secondary, which may in turn be surrounded by a tertiary, invagination.

Intussusception is ordinarily classified in accordance with the anatomic features. Perrin and Lindsay[3] suggested that the classification be in two groups: (1) ileocecal and (2) ileocolic.

In the first variety the ileocecal valve forms the peak of the intussusception, while in the second, the invagination begins in the ileum adjacent to the valve and passes through it into the cecum.

There are, in reality, three forms:

1. Enteric (10 to 15 per cent), in which the small intestine invaginates. This form is prevalent in older children and adults. It is usually of organic causation. In the ileo-ileal variety only the small intestine is involved; it may progress to become an ileo-ileocolic variety, ordinarily discoverable in children.

2. Colic (5 to 10 per cent), in which the colon ensheaths (therefore, colo-colic). This variety occurs in elderly persons. A not infrequent cause is a polypoid carcinoma or a benign polypoid growth.

3. Enterocolic (75 to 80 per cent), in which the ileum invaginates into the colon. This is common in infants. In this category there are three distinct subvarieties:

(a) Ileocecal, the ileum passing into the colon, with the ileocecal valve as the fixed vertex.

(b) Ileocolic, beginning in the terminal ileum and then progressing into the colon.

(c) Enteric ileocecal, starting as an enteric intussusception, becoming wedged in the ileocecal valve, which it pushes forward, and then advancing as an ileocecal intussusception, with the valve at its summit.

Retrograde Intussusception.—A retrograde bowel ensheathment is said to occur only in the small intestine at the time of death. It is, however, seen occasionally during life and may occur at a gastroenterostomy opening.

Jejunogastric Intussusception.—When a gastroenterostomy has both an afferent and efferent jejunal loop, the following three varieties of jejunogastric intussusception may, on rare occasions, occur as complications within a few days or years after the anastomosis:

1. The afferent loop solely may intussuscept into the stomach.

2. The efferent loop may retrograde and stop short of or pass through the gastroenterostomy stoma.

3. The afferent and efferent loops may intussuscept into the stomach.

Etiology of Jejunogastric Intussusception.—Pregnancy at one time was considered an embryologic factor, but the majority of reported cases of jejunogastric intussusception had no association with that condition and many have been described as occurring in men.

In most instances the afferent and efferent intestinal loops passed into the stomach.

Symptoms of Intussusception.—In adults the symptoms of intussusception are not as prominent as in infants. They are vague in most instances, with

signs of mild recurrent obstructions associated with symptoms of the provoking cause.

There are coliclike abdominal pains, nausea, vomiting, obstipation, and bloody stools.

A mass in the abdomen is commonly not palpable.

In jejunal intussusception, symptoms usually intermittent may be present for months or years. There is always a history of upper abdominal pain, especially after meals. Nausea and vomiting are common. Blood in the stools is not as common in jejunal intussusception.

The abdomen is not distended early, but becomes manifestly so as the obstruction progresses.

On auscultation exaggerated peristaltic sounds may be heard, until complete obstruction, when one elicits the significant silent abdomen.

Palpation usually reveals tenderness and rigidity over the involved area. (The first may be absent as well as the second, due to the fact that the parietal peritoneum is not irritated and that protection is afforded by the ensheathing intussuscipiens.)

A tumor mass may be felt, with or without anesthesia. It is sausage shaped and lies transversely across and subjacent to the abdominal wall.

In early cases a barium enema may be given at low pressure and an attempt should be made to reduce the mass under fluoroscopic control. This is often surprisingly successful in the colic or enterocolic varieties.

It is almost impossible to reduce the enteric variety merely by rectal injection.

Diagnosis and Differential Diagnosis.—The differential diagnosis is often confusing, with the exception of those instances in which a palpable tumor is present. Here the sudden concurrence of an abdominal tumor, usually in the right lower quadrant, with the symptoms of intestinal obstruction, is suggestive but not absolutely indicative of intussusception. A large bowel malignancy, for example, may simulate intussusception.

The diagnosis is mainly roentgenographic or surgical.

Diagnosis of Jejunogastric Intussusception.—If the afferent loop is intussuscepted, the patient will as a rule complain of (1) postprandial epigastric pain with colicky exacerbations and (2) bilious vomiting.

The roentgenograms in most instances will reveal a distended afferent loop. The opaque meal will demonstrate a rounded filling defect in the stomach (due to the intussusception) upon which a relief pattern, the jejunal plicae, will be observable.

If the efferent loop takes a retrograde intussusceptal course, an acute high small bowel obstruction results with consequent colicky abdominal pain and the vomiting of gastric, then bilious, and finally intestinal contents.

If the afferent and efferent loops intussuscept into the stomach, an acute, high obstruction obviously results.

The frequent difficult differential diagnosis is jejunogastric intussusception is due to the fact that the symptoms are interpreted as the result of other possible complications of the gastroenterostomy—stomal inflammation or ulceration, for example, or complications due to the primary causative factor, a leaking perforative or bleeding ulcer.

The differential diagnosis in intussusception must likewise be made from (1) Henoch's purpura, (2) acute enterocolitis, (3) simple colic, and (4) tuberculous mesenteric glands.

The Roentgenographic Examination.—The barium enema and examination of the abdomen without opaque media are used to diagnose intussusception. The opaque or barium enema is preferred by many roentgenologists.

When an acute abdominal condition is present or suggested by the symptoms, an examination without opaque media is of course preferable.

In subacute and chronic intussusception in adults it will be found that the combined contrast meal and enema examinations will prove most helpful in diagnosis.

The roentgenograms will reveal (1) dilatation of the gut above the invaginated part, (2) sudden narrowing over a variable length of gut, which is the central cylinder of the intussusception, and (3) various degrees of the "ensheathment sign."

The plain film manifestations of enterocolic intussusception are (1) absence of the mottled appearance of the cecum, due to slight gas and fecal content, (2) collection of gas-distended small gut loops in the region normally occupied by the cecum, (3) homogeneous opacity of the intussusceptum, (4) peripheral concentric "ringing" by translucent gas, (5) conical stenosis of the small gut as it enters the intussusception, and (6) direct visualization of the tumor provoking the intussusception.

Treatment.—

Palliative: Palliative treatment is justifiable only under the most exceptional circumstances. An early intussusception can sometimes be completely reduced with a pressure enema of water or air. This method is dangerous and is better employed in infants and older children.

Surgical: An immediate surgical operation should be performed as soon as the diagnosis is made. Ether anesthesia is best avoided. Spinal anesthesia is preferred. A one-stage operation may be performed.

When a hot pack fails to effect reduction, an attempt should be made, according to Cope,[4] to insert a finger into the neck of the intussusception (between the entering and returning layers) and to sweep it around in this space; the adhesions are thus broken down and reduction facilitated. If this maneuver proves ineffective, a rubber-covered forceps is placed in the neck as far as it is safely possible and the blades are opened in several places. This procedure serves the dual purpose of dilating the neck and of breaking down recent adhesions.

If the aforementioned methods fail, the neck should be divided for an inch or more along the antimesenteric border with a pair of forceps. The incision is closed transversely to avoid narrowing of the lumen of the gut.

If the large intestine is the intussusception site, graded operations are preferable. Attempts at reduction and pressure should be made over the peak of the intussusception, with simultaneous light traction on the proximal free bowel.

In some cases of irreducible, uncomplicated intussusceptions a short-circuiting operation may be performed. If the intestine is irreducible and gangrenous, one of the following expedients may be tried:

(1) Resection, with end-to-end, side-to-side, or end-to-side anastomosis.

(2) Jesset's operation—removal, solely, of the invaginated part of the bowel through the encompassing part of the intestine. After the incision in the bowel is made, the invaginated part is withdrawn in a downward direction as far as possible and cut away. Circular suturing closes the cut margins of the amputated, gangrenous bowel. The ensheathing part of the bowel is

then approximated and united by a through-and-through continuous suture whereon a Lembert stitch is placed. A few interrupted sutures close the neck of the intussusception.

The circumjacent and contiguous parts are next explored to discover a tumor. If it is not found at the apex of the intussusception, the bowel should be further examined, both proximally and distally.

The Management of Prolapsed Sigmoidal Intussusception.—The patient is placed in steep Trendelenburg position and a spinal anesthesia is given.

A firm rubber tube, about one inch in diameter, is passed through the lumen of the invagination and the prolapsed sigmoid to a level a few inches beyond the site of involvement; a tight rubber band is now passed over the invaginated sigmoid to the level of origin of the invagination. The intussuscepted sigmoid subsequently sloughs off because of the two constricting devices and an anastomosis is thus established.

Treatment of Jejunogastric Intussusception.—The treatment of jejunogastric intussusception is always surgical. If the afferent loop is involved, reduction is effected by slight traction. A recurrence is prevented either by anchoring the afferent loop to the stomach or to the transverse mesocolon or by undoing the gastroenterostomy. Sometimes a gastrectomy, in part, including resection of the anastomosis, may be deemed advisable.

If the efferent loop is entangled, reduction by traction may be found adequate, but in delayed cases, with gangrene, resection, obviously, must be carried out.

When both intestinal loops are involved, reduction is by traction.

Prognosis.—Sometimes there is a spontaneous reduction of an acute intussusception of the recurrent variety. The prognosis, when palliative measures prove ineffective, depends, of course, upon early and quick surgical care.

CASE REPORTS

CASE 1.—S. S., a 49-year-old white man, a taxicab driver, was admitted to the hospital in February, 1930, complaining of cramplike circumumbilical pains, nausea, and almost complete obstipation of one week's duration. He had taken several enemas without result. Ten years previously he had had an appendectomy and gastric operation for ulcer; the type of operation was unknown.

Present Illness.—The day previous to admission, the patient's pains became more severe and he vomitted a black-colored material on several occasions. Shortly after admission he vomited foul, fecal-smelling material.

Physical Examination.—On physical examination, he presented a picture of profound shock. He was extremely pallid and peaked and appeared in extremis. General physical examination was negative with the exception of the abdomen. The latter presented a right lower rectus and left upper rectus scar and was not distended. Tenderness was present all over the abdomen and was most marked in the right lower quadrant where marked rebound tenderness was present. The temperature was 101.4° F., pulse, 120; blood pressure, 88/70; white blood cells, 15,400 with 86 per cent polymorphonuclear cells. The urine contained a faint trace of albumin with many hyaline and granular casts.

Surgical Procedure.—In view of the patient's precarious condition, no extensive operative procedure was contemplated. That he was suffering from an acute intestinal obstruction was obvious; a scaphoid abdomen was present and there were fecal vomiting and almost no pulse. Nevertheless his poor condition demanded an emergency procedure. The origin of the obstruction was believed to be traceable to adhesions from his previous laparotomies. The left upper abdomen was opened through a very small incision measuring about two and one-half inches in length, parallel to the rib margin, the first distended loop of jejunum secured, and a Senn jejunostomy performed with marsupialization of the bowel to the parietal peritoneum. A large quantity of free serous fluid was noted in the abdomen. One hour after the operation the patient showed marvelous improvement. He felt better and the

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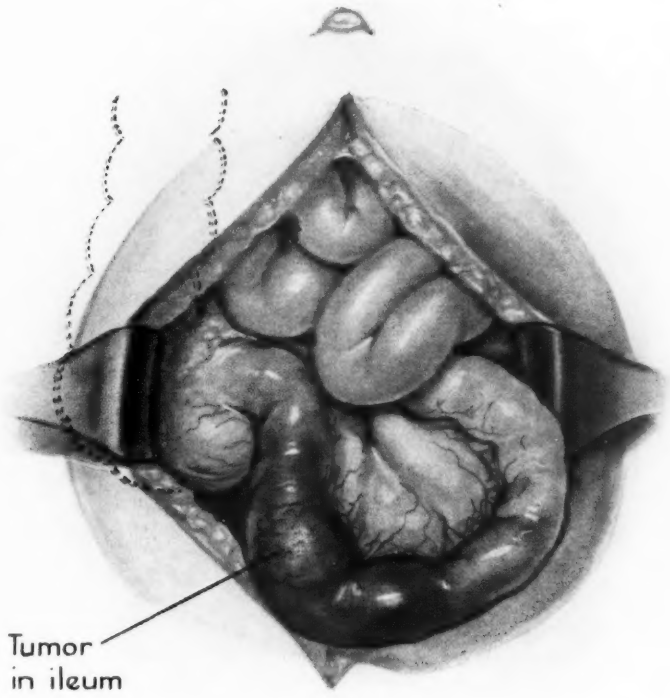

Fig. 1 (Case 1).—Adenomatous polyp of ileum.

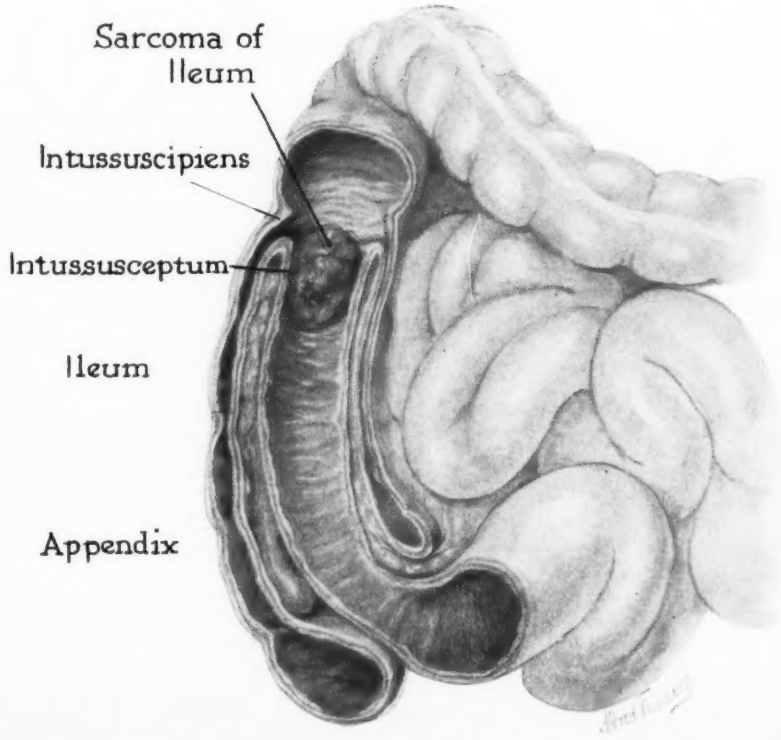

Fig. 2 (Cases 2 and 6).—Sarcoma of intussusceptum (ileum).

pulse was of good quality. There was no vomiting or nausea. Postoperatively as well as preoperatively the patient was given large quantities of saline by hypodermoclysis and intravenously. Drainage was rather profuse through the jejunostomy tube, moderate quantities of food were retained by mouth, and there were occasional small bowel movements and flatus passed. The cramplike abdominal pains persisted, however, at first less severe but gradually increasing in severity. Twelve days postoperatively the abdomen was opened again, through a lower midline incision. The small intestine was markedly distended and there was found a large intussusception of the ileum through the ileocecal valve into the cecum and ascending colon, involving about 25 cm. of intestine. The intussusception was easily reduced by slowly milking out the intussusceptum in the accepted manner. The ileum was found to be almost black, markedly inflamed, edematous, and lusterless and showed evidence of beginning gangrene. A few inches from the ileocecal valve was felt a firm mass inside the lumen of the ileum close to the apex of the intussusception. The ileum with the encircling mass was brought outside the abdominal wall and a first stage Mikulicz operation performed. In this case an artificial anus was established through the small bowel. Due consideration and thought were given to the resultant liquid continuous fecal discharge and possible progressive malnutrition. However, the patient's condition, together with the pathologic findings, required heroic yet unorthodox measures. Nevertheless, the end justified the means. Drainage through the previous jejunostomy was maintained through a catheter. Three days later, that portion of ileum which had previously been brought outside the abdomen was resected to within a short distance from the abdominal wall by cautery. Nine days later a spur clamp was applied in the usual manner and twenty-five days following the first stage the continuity of the ileum was re-established. The postoperative course was entirely uneventful. Three days after the final closure the patient had his first normal bowel movement, and two weeks following, was discharged.

Pathology.—The resected intestine was 19 cm. in length. At operation there was found a cauliflower growth within the lumen of the gut, about 4 cm. in diameter, attached to the intestinal wall by a short pedicle. About 2 cm. proximal to this was found a second polypoid mass about 3 cm. in diameter, discolored and red, with a smooth surface. This growth could not be felt at the time of operation.

The surrounding intestine was quite edematous. Microscopic examination showed the intestinal mucosa to be the seat of a localized area of hyperplasia of the glands from which there extended a mass of mucoid substance in which were found strands of deep-staining epithelial cells, distorted in outline. Although the superficial portion of the nodule showed no tendency to alveolar arrangement of the epithelial cells, the portion adjacent to the adjoining normal mucosa was definitely adenomatous in character. There was an associated polymorphonuclear cell and mononuclear cell infiltration with numerous plasma cells found at the base of the nodule in the surrounding intestinal wall. The edema was limited to the superficial portion of the mucosa. The pathologist's diagnosis was benign adenomatous polyp with mucoid degeneration.[4]

CASE 2.—G. H., a 68-year-old white man, a painter, was admitted to the hospital Oct. 25, 1939. His chief complaint was paraumbilical pain radiating to the right lower abdominal quadrant.

Family and Past Personal History.—Histories were irrelevant.

Present Illness.—The present illness began about one month previously. The patient experienced paraumbilical, cramplike pain radiating to the right lower quadrant. There was no nausea or vomiting. The attacks lasted several minutes and gradually disappeared. For four or five days prior to admission pain was almost continuous. The bowel movements were regular and there was no melena.

Physical Examination.—A complete physical examination disclosed no abnormalities, with the exception of a tender mass palpable in the right lower abdominal quadrant, adjacent to the right side of the umbilicus. The mass was not easily displaceable. The blood pressure was 142/100; hemoglobin, 85 per cent; red blood cells, 4,700,000; white blood cells, 6,000; polymorphonuclears, 80 per cent; no basophilic stippling of red blood cells.

Operation.—October 27, two days after admission, an exploratory laparotomy was performed, through a right rectus incision, under spinal anesthesia (procaine).

Surgical Data.—An intussusception of the lower ileum into the cecum and ascending colon was discovered, with some degree of inflammation about the involved loops. The intussusception was released with gentle pressure from the intussuscipiens; this maneuver exposed a firm, indurated ileal tumor on the antimesenteric border, the size approximating a fifty-cent coin.

Impression.—The impression was sarcoma of the ileum.

Surgical Procedure.—The lower ileum for an extent of six inches and the lower ascending colon and cecum were mobilized and exteriorized (Lahey technique). The protruding loop was then resected, forming a double-barreled enterostomy. The abdomen was closed in layers and petroleum jelly gauze was inserted into the wound around the exposed intestine.

Pathologic Report.—A small-cell sarcoma was found infiltrating the musclaris and serosa.

Postsurgical Therapy.—Hypodermoclyses of glucose in saline solution were given. Drainage through the colostomy was profuse. The general condition of patient was satisfactory. November 5 (ninth postsurgical day), a spur clamp was applied; this fell spontaneously out of the wound November 11 (six days after application).

Examination several days later revealed presence of a small spur, and a clamp was reapplied November 20; it fell out again November 23.

The patient was prepared for a third stage Mikulicz operation, or closure of the enterostomy, which was carried out, under spinal anesthesia, November 24.

Postsurgical Course.—At first the postsurgical course was stormy. The pulse was weak, thin, and rapid. The blood pressure was 78/60. Perspiration was profuse and the extremities cold. The patient was placed in the Trendelenburg position and intravenous infusion of 10 per cent glucose was slowly administered. There was good response in two hours and the general condition of the patient gradually improved. A small amount of drainage, fecal in character, appeared through the abdominal wound but stopped in a week.

The patient was well four years after the operation.

CASE 3.—A. T., a white housewife aged 36 years, was admitted to the hospital Dec. 16, 1933. She complained of intermittent abdominal cramps, loss of weight, and weakness of one and one-half years' duration. The cramps were at first generalized, then of increased severity, localizing around the umbilicus; they gradually diminished in severity. Relief frequently was obtained by passage of flatus. The last, most severe attack began one week prior to admission and was accompanied by nausea and a vomiting spell. The weight loss was 80 pounds in eighteen months.

On admission the patient was restless, moving from side to side, with thighs flexed. The temperature was 103° F.; pulse, 114; and blood pressure, 120/80.

Physical Examination.—The abdomen was moderately spastic. A slightly tender mass, about 7 cm. in diameter, was palpable to right side of umbilicus.

Surgical Procedure.—The following day the abdomen was explored through a right rectus incision. There was serosanguineous fluid in the peritoneal cavity, and an irreducible intussusception of the ileum into the colon as far as the distal third of the transverse part was discovered. There were involved loops of intestine and mesentery, extremely congested, edematous, and adherent. The affected bowel was mobilized, brought to the surface, and resected. The wound was closed about the two intestinal barrels. The excised mass of intestine, 34 cm. long and 8 cm. wide, included cecum, appendix, and about 30 cm. of invaginated ileum.

Second Postsurgical Day.—A rubber drainage tube was inserted into the proximal opening. The abdomen was soft. The temperature varied between 101 and 102° F.; pulse, 100 to 120.

A spur clamp was inserted December 26 (nine days after enterocolectomy) and was removed December 31.

Anastomosis was completed by a third stage Mikulicz procedure Jan. 4, 1934. Four days later the patient had the first spontaneous bowel movement. Small fecal discharge appeared in the abdominal wound and continued less than a week.

The patient was discharged from the hospital Jan. 15, 1934, in good general condition.

Pathology.—Intussusception with areas of necrosis in mucosa and muscularis of ileum and colon was found. There was no evidence of tumor formation.

CASE 4.—H. B., a white man, aged 33 years, was admitted to the hospital Feb. 12, 1936. His chief complaint was spasmodic abdominal pain of six months' duration recurring at intervals and radiating to right and lower abdominal quadrants. The pain at first occurred daily; at the time of admission it occurred on alternate days but was of greater intensity. There was no relationship to food intake. There was some loss of weight and the appetite was poor due to pain.

Physical Examination.—A slightly movable mass was found in left upper abdominal quadrant.

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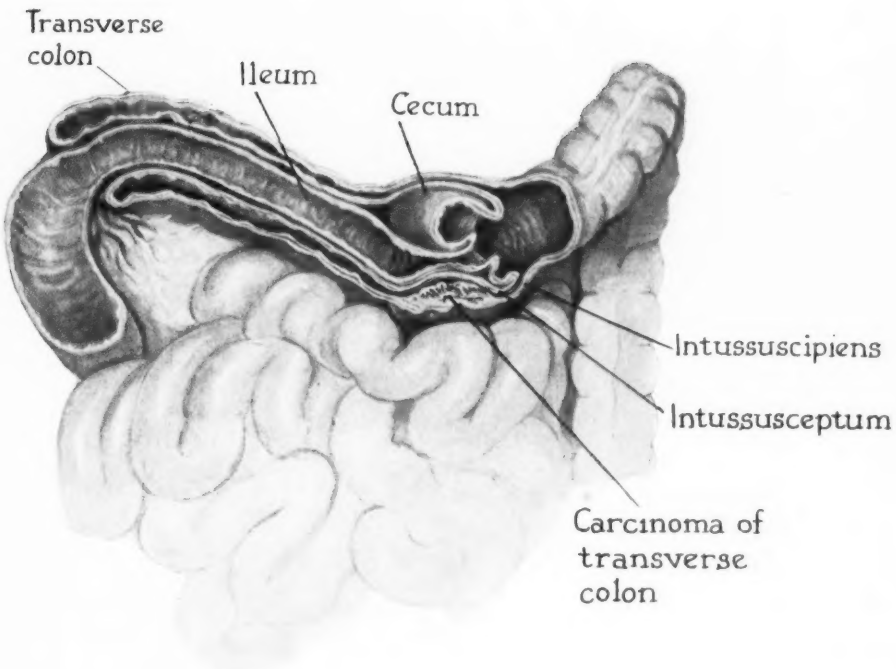

Fig. 3 (Case 4).—Carcinoma of intussuscipiens.

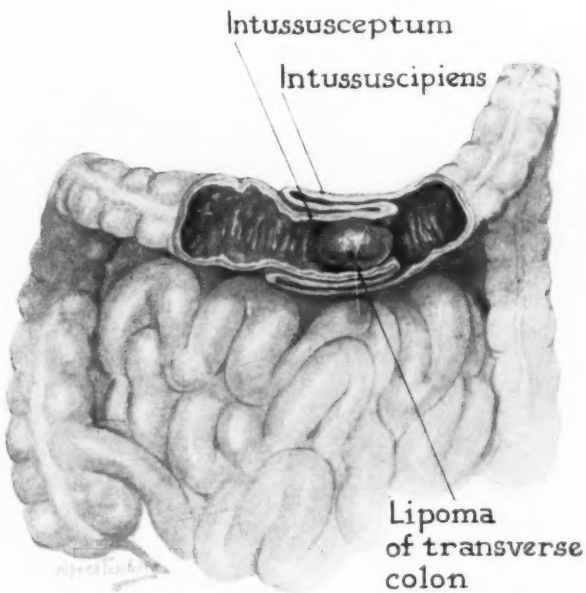

Fig. 4 (Case 5).—Submucous lipoma of intussusceptum.

Operation.—An operation was performed Feb. 14, 1936, using an upper left rectus incision.

Surgical Data.—A large mass, with evidence of subacute or chronic inflammation, involved a large extent of the transverse colon. The cecum and appendix were missing. The lower ileum, with cecum and ascending colon, were found intussuscepted into the transverse colon up to the splenic flexure. Intussuscipiens, with lost luster, showed extreme hypertrophy. The apex of the intussusception (situated near the splenic flexure), on palpation, gave the impression of a mass. There were edema and congestion of the mesentery of the transverse colon and small amount of fluid present in the peritoneal cavity.

Surgical Procedure.—The mass was reduced by careful pressure on the intussuscipiens, distal to apex of intussusceptum, thereby freeing the bowel. Induration and apparent loss of viability of cecum and ascending colon suggested the removal of this part of the intestine, including the lower ileum. The condition of the patient made it expedient to carry out ileocolectomy, in part, by a multiple-stage procedure. The lower six inches of ileum and entire ascending colon, and part of transverse colon, were brought into the wound in the manner of the Mikulicz operation.

Pathology.—Carcinoma of splenic flexure of colon was found.

Comment.—This case was of special interest because there was a carcinoma in the intussuscipiens and not in the intussusceptum.

The patient made uneventful recovery.

CASE 5.—J. A., a 39-year-old white man, a plumber, was admitted to the hospital Feb. 8, 1941. His chief complaint was abdominal cramps of three weeks' duration, with nausea and gradual loss of weight.

Physical Examination.—A mass in the left upper abdominal quadrant was discovered.

Roentgenographic Examination.—There was an obstruction near the splenic flexure.

Surgical Data.—A first stage Mikulicz operation was performed Feb. 16, 1941. Intussusception of the ascending colon into the transverse colon as far as the splenic flexure was found. There was extreme edema of walls of transverse colon, as well as of gastrocolic omentum, with moderate thrombosis of gastrocolic vessels.

Surgical Procedure.—The intussusception was reduced, revealing extreme induration and swelling at the apex of the intussusception. The necrotic condition of the bowel required a multiple-stage resection of the diseased part of the transverse colon. The transverse colon was exteriorized up to the splenic flexure and removed by cautery (Feb. 20, 1941) four days after the first stage procedure. A spur clamp was applied immediately, but it was found necessary to apply another spur clamp six days later in order to remove the residual spur about one inch in length. Four days after the first stage procedure a second one was carried out, with removal of exteriorized loop. A spur clamp was applied March 15, 1941. March 22, 1941 (five weeks after the first procedure), continuity of the bowel was re-established. Union was per primam, except for a small serous collection subcutaneously at the upper angle of the wound. The patient recovered.

Pathology.—A submucous lipoma of the mid-part of the transverse colon was found.

CASE 6.—H. H., a 60-year-old white man, was admitted to the hospital March 16, 1942, with a history of repeated attacks of abdominal cramps radiating to right loin. He complained of constipation, slight weight loss during the previous two months, and anorexia.

Present Illness.—For the sixteen hours prior to admission the patient had had severe abdominal pain, cramplike in character, especially on the right side. He had had no bowel movement for thirty-six hours, but in the few hours before admission had noticed a slight sanguineous mucoid discharge from the anus.

Physical Examination.—A slightly distended abdomen with a moderately tender, slightly movable, elongated mass in right mid-abdomen was disclosed.

Surgical Data.—A right rectus incision was made March 16, 1942.

Findings.—An ileocolic intussusception with marked discoloration, edema, and dullness of ascending colon was found. The reduced intussuscipien was almost black, and at a point four inches proximal to the ileocecal valve there was a firm egg-shaped mass about three-fourths by one and one-half inches, apparently adherent to the anterior ileal wall. There was a moderate free serosanguineous fluid in the peritoneal cavity. The mesentery of the lower ileum showed several thrombosed vessels.

Surgical Procedure.—In view of the pathology found, it was deemed advisable to do a side-to-side ileotransverse colostomy and a primary resection of the lower ileum and ascending colon.

The patient made an uneventful recovery, and one year later was entirely symptom free.

Pathology.—Sarcoma of lower ileum was found.

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AN IMPROVED METHOD FOR EXTRAPERITONEAL CLOSURE OF COLOSTOMY

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IN ORDER for the surgical closure of a colostomy to be successful, it is necessary to overcome many obstacles which are not present in most other operative procedures. The entire operation for re-establishing the continuity of the bowel must be performed in an infected field and, therefore, healing must take place in the presence of inflammation in an individual whose resistance has been lowered by the primary lesion for which the colostomy was originally performed. All of these factors are detrimental to firm wound healing and favor the development of complications.

Therefore, the purposes of this paper are to present an improved method for extraperitoneal closure of colostomy and to demonstrate its advantages in an analysis of seventy-five consecutive cases over a ten-year period.

TECHNIQUE

The operative steps which are preliminary to the actual closure of the colostomy, as illustrated in Fig. 1 (*A* to *F*), are as follows:

An elliptical skin incision is made, longitudinally around the colostomy down to the anterior rectus sheath, leaving a narrow rim of skin attached to the stoma for traction (*A* and *B*). The subcutaneous tissue is carefully dissected from the anterior rectus sheath and bowel wall, leaving a one-inch margin of cleaned fascia around the colostomy (*C* and *D*). A circumferential incision is then made through the anterior rectus sheath leaving a one-fourth inch rim of fascia attached to the bowel which will be subsequently utilized as an extra layer in the closure (*E*). The excess bowel and attached rim of skin are now removed (*F*).

The operative steps involved in the actual closure of the colostomy, as illustrated in Fig. 1 (*G* to *L*), are as follows:

Closure of the bowel in its transverse axis is accomplished by the use of the Connell stitch reinforced by the Lembert stitch (*G* and *H*). The circumferential rim of fascia

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(described previously and illustrated in Fig. 1*E*) is approximated longitudinally by a continuous suture (*I*). This is the most important step because it provides an additional firm, supportive layer for the closure and also relieves tension on the intestinal suture lines beneath, which favors rapid healing and thereby reduces the incidence of local complications such as fecal fistula, wound infection, dehiscence, and hernia. Tension sutures are then inserted at one-inch intervals through the skin, passing beneath the margins of the anterior rectus sheath (*J*). The anterior rectus sheath is approximated by mattress sutures so spaced as to allow for drainage between them (*K*). A Penrose drain is placed in the depth of the wound and protrudes from both ends of the incision, after which the tension sutures are tied to approximate the skin and subcutaneous tissue (*L*).

The average operating time for this procedure was found to be thirty-five minutes.

ANALYSIS OF SEVENTY-FIVE CASES

In this series (Table I) it was found that the average age was 51 years and that females predominated by 12 per cent. Furthermore, of the lesions necessitating colostomy, the malignant predominated over the benign in the ratio of 2.5:1.

TABLE I. INCIDENCE OF SEVENTY-FIVE COLOSTOMY CLOSURES

AGE	SEX	NO.	%	PATHOLOGIC LESION	NO.	%
Extremes	Males	33	44	Malignant	53	70.6
23-75 yr.	Females	42	56	Nonmalignant	22	29.4
Average				Polyp	5	
51 yr.				Diverticulitis	6	
				Chronic volvulus	4	
				Stricture	4	
				Ulcerative colitis	2	
				Regional enteritis	1	

In an analysis of the relative time factors incident to closure of these colostomies at their various locations (Table II), it was found that the average interval between the performing of the colostomy and its closure was twelve weeks, but that this varied with the pathologic lesion so that it was six weeks in the group of malignancies and twenty-six weeks in the benign. This time factor was increased in the latter (enumerated in Table I) because of the necessity of specific interval therapy before the colostomy was closed, and was found to be fifty-one weeks in diverticulitis and approximately fifteen weeks in each of the remaining lesions in this group. It was further revealed that the minimum time interval between the colostomy and its closure was three weeks, allowing ten days before the spur was crushed and another ten days before the colostomy was closed. This occurred only in the group of malignancies.

Following colostomy closure, in all locations it was found that normal stools appeared after an average of five days and that the average hospital stay was three weeks. A significant finding was that the period required for complete healing of the operative site averaged four weeks. Further analysis revealed

TABLE II. TIME FACTORS RELATED TO COLOSTOMY CLOSURES

LOCATION	NO. OF CASES	PREPARATION PERIOD: TIME BETWEEN COLOSTOMY AND SPUR CRUSH (WEEKS)	PREPARATION PERIOD: TIME BETWEEN SPUR CRUSH AND CLOSURE (WEEKS)	CONVALESCENT PERIOD: PERIOD BEFORE NORMAL STOOLS (DAYS)	CONVALESCENT PERIOD: HOSPITAL STAY AFTER CLOSURE (WEEKS)	CONVALESCENT PERIOD: PERIOD FOR COMPLETE HEALING (WEEKS)
Sigmoid	31	9	5	5	3	4
Transverse	29	5	4	5	3	5
Descending	10	9	5	4	3	4
Cecum	5	-	-	5	3	2
Average		7	5	5	3	4

that 94.6 per cent (seventy-one cases) were healed within five weeks, of which 66.6 per cent (fifty cases) required two weeks, 17.4 per cent (thirteen cases) required three weeks, and only 10.6 per cent (eight cases) required five weeks.

The results in this series are shown in Table III. Primary healing occurred in sixty-one cases (81.3 per cent). There were ten instances (13.3 per cent) of fecal drainage. Of these, six healed without additional surgery within five

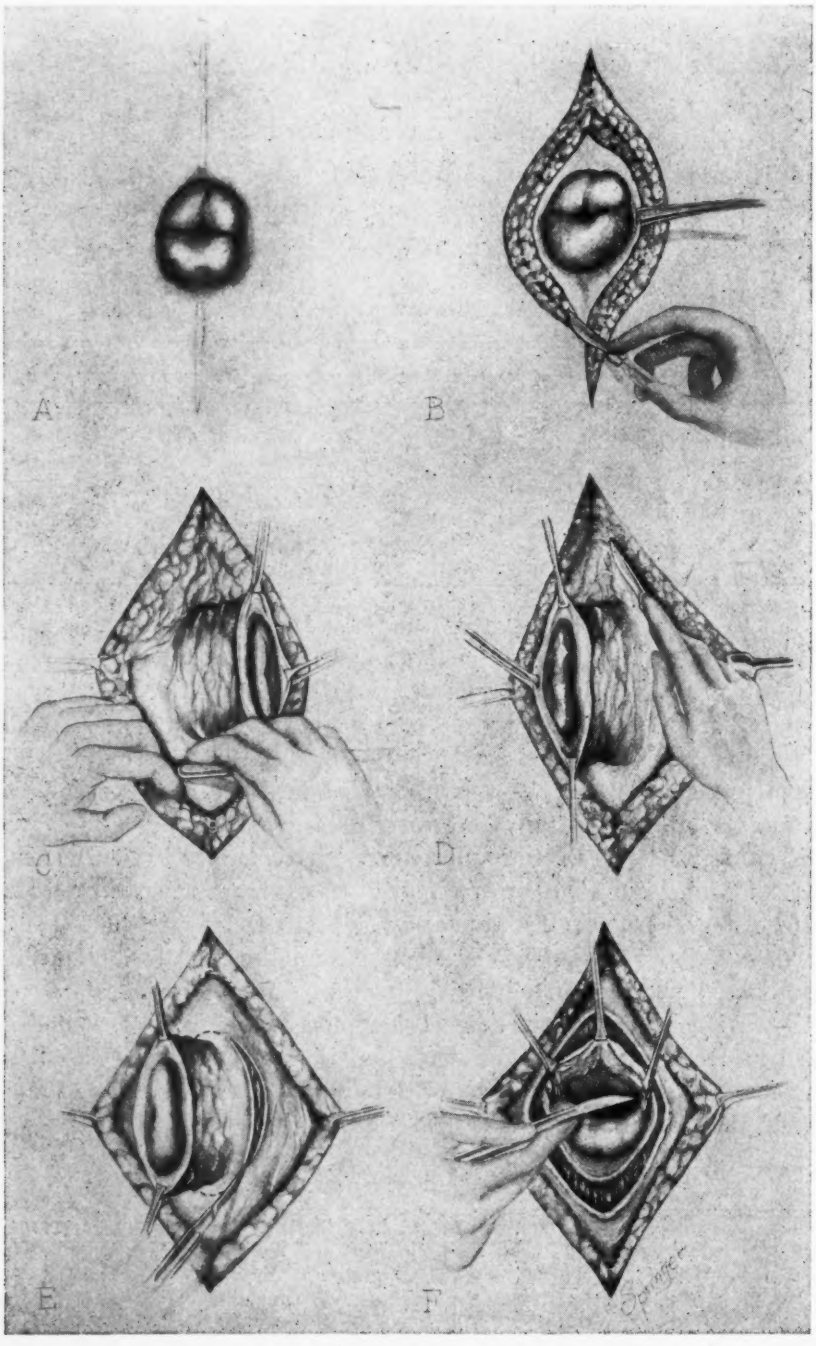

Fig. 1 *A* to *F*.—Operative steps preliminary to the actual closure of the colostomy. *A*, Colostomy with healed original incision. *B*, Elliptical incision around colostomy down to anterior rectus sheath, leaving narrow rim of skin attached. *C* and *D*, Dissection of subcutaneous tissue from anterior rectus sheath and bowel wall leaving one inch margin of cleared fascia around the colostomy. *E*, Circumferential incision through anterior rectus sheath leaving a one-fourth inch rim of fascia attached to bowel. *F*, Excision of rim of skin and adjacent excess mucous membrane from the colostomy stoma.

weeks, and two in six months. One persisted for one year, at which time the tract was dilated sufficiently to permit further crushing of the spur which was followed by complete healing in seven weeks. The remaining case, of five months' duration, necessitated secondary operative closure after the residual spur was crushed; healing then took place in three weeks.

Although some degree of inflammatory reaction occurred in all the wounds in this series, in only four cases (5.3 per cent) did it extend beyond the usual

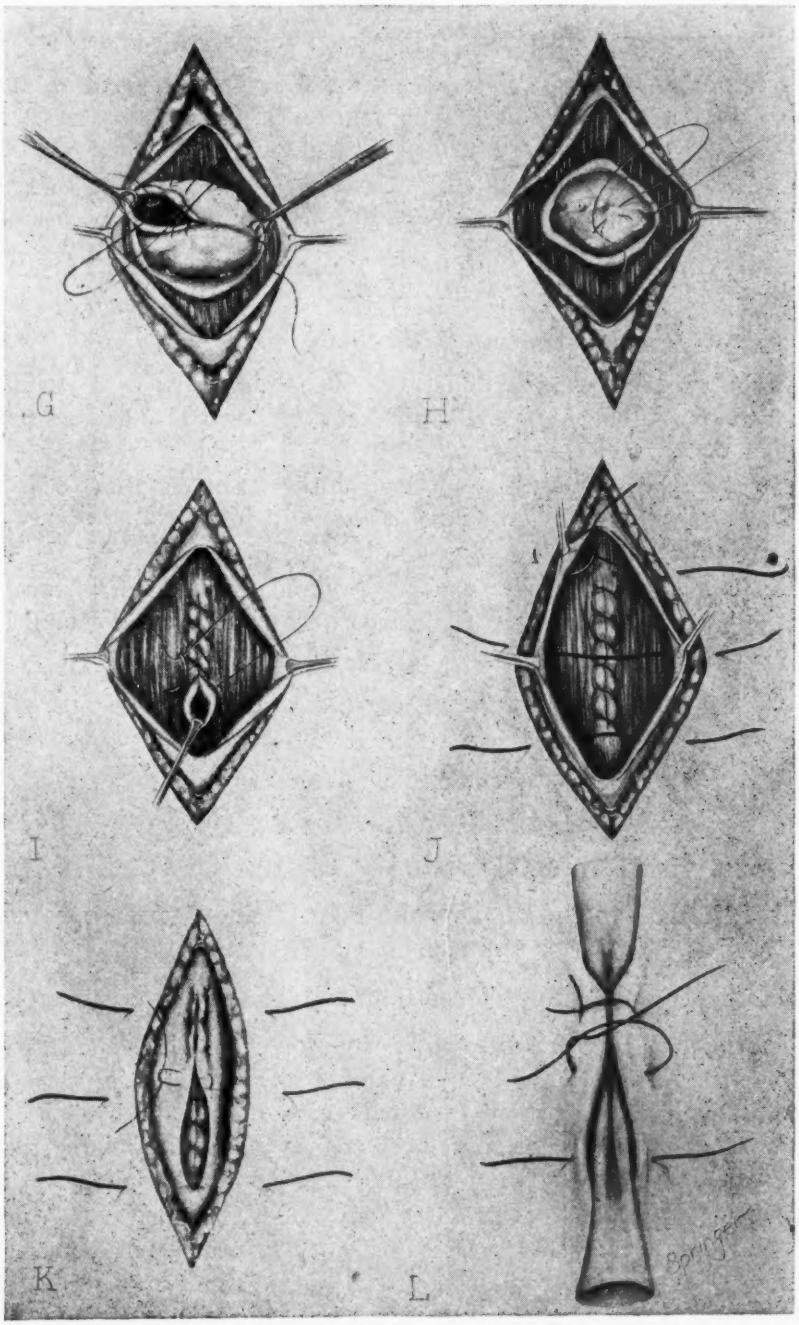

Fig. 1 *G* to *L*.—Operative steps in the actual closure of the colostomy. *G* and *H*, Closure of bowel in its transverse axis by the Connell stitch overlaid by the Lembert stitch. *I*, Circumferential rim of rectus fascia approximated longitudinally by a continuous suture. *J*, Tension sutures inserted through skin, passing beneath margins of anterior rectus sheath. *K*, Anterior rectus sheath closed by mattress sutures. *L*, Tension sutures approximating skin and subcutaneous tissue; Penrose drain protruding from both ends of incision.

TABLE III. RESULTS IN SEVENTY-FIVE COLOSTOMY CLOSURES

			CECUM	TRANSVERSE	DESCENDING	SIGMOID
	NO.	%	NO.	NO.	NO.	NO.
Primary healing	61	81.3	5	22	9	25
Fecal drainage with delayed healing	8	10.6		4	1	3
Fecal drainage requiring secondary spur crush	1	1.3				1
Fecal drainage requiring secondary spur crush and operative closure	1	1.3		1		
Wound cellulitis without fecal drainage	4	5.3		2		2

five- to seven-day period, and in no instance was surgical interference necessary. The average time for the cellulitis to subside in these four cases was three weeks.

There were no deaths in this series attributable to complications resulting from closure of the colostomy. However, one death occurred on the twenty-sixth postoperative day due to uncontrollable hemorrhage from a coexisting carcinoma of the stomach as proved by autopsy.

SUMMARY AND CONCLUSIONS

A technique for extraperitoneal closure of colostomy, as employed in seventy-five consecutive cases, is described and illustrated. The keystone of this method is a fascial rim of external rectus sheath which is left adherent to the bowel wall and is utilized as an extra layer in the closure. This added, firm support relieves tension on, and enhances healing of, the intestinal suture lines beneath, thereby minimizing complications.

In an analysis of this series we have found this method to have the advantages of being safe and easily performed with a minimum of complications.

DYNAMICS OF BILIARY DRAINAGE

ITS RELATION TO CHOLANGITIS AND PANCREATITIS FROM STRICTURE OF THE AMPULLA OF VATER

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THE purpose of this presentation is twofold. First, it demonstrates how a minor stricture of the ampulla of Vater can lead to a series of pathologico-physiologic disturbances involving the pancreatic and biliary systems. Second, it affords an excellent opportunity for a step-by step study of the therapeutic measures based on physiology necessary to restore conditions to normal.

Following surgical removal of the gall bladder, the patient's condition is very often no better than before the operation. The poor results reported following biliary surgery can be explained on two bases. First, an incorrect diagnosis may have been made preoperatively. A gall bladder removed for complaints based on pathology in another organ or on psychoneurosis can be of no benefit

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to the patient. Furthermore, a young woman from whom a normally functioning gall bladder has been removed is left with a biliary tract which has not had time to accustom itself to the absence of the pressure-regulating mechanism of the gall bladder. A whole series of symptoms may develop in connection with the biliary system which the patient did not have prior to operation. Second, even after a diseased gall bladder is removed, continued symptoms may follow because of inability of the surgeon to pay attention to the physiology of the biliary and pancreatic systems. The actual cholecystectomy is only a part of the treatment for the patient's condition. The liver, pancreas, and extrahepatic biliary tract must be carefully studied for evidence of pathology and treated accordingly.

Following removal of the gall bladder, the postoperative care of the patient begins in the operating room. One of the most important things in this connection is the T-tube drainage of the common bile duct. The indications for opening the common bile duct at the time of operation have been clearly defined by numerous writers and are generally accepted. Some of these indications are: a history of repeated chills or jaundice, the presence of palpable stones in the common bile duct, pancreatitis, a markedly dilated duct. When the duct has been opened and a thorough search for stones made, a T tube should always be inserted for prolonged postoperative biliary drainage.

Besides its therapeutic benefits the T tube serves as a means for studying the biliary and pancreatic systems. T-tube studies are usually begun two weeks after the operation. These consist of pressure and roentgenologic studies. In a previous communication[2] it was shown that duodenal spasm is a very important factor in preventing bile from running into the duodenum. The resulting back pressure in the common duct often leads to pain. This duodenal spasm was shown to be aggravated by morphine and to be completely relieved by amyl nitrite and nitroglycerin. The case referred to in this report is not, however, one in which duodenal spasm is a prime factor. It is a case of pancreatitis, cholangitis, and cholecystitis, all believed to be due to a stricture of the ampulla of Vater at the duodenal mucosa.

RESTING INTRABILIARY PRESSURE

The method of intrabiliary pressure studies has been previously reported.[1] The apparatus essentially consists of a spinal fluid manometer connected to the T tube in the patient's bile duct. Between the two is a glass Y tube through which saline solution may be added to the apparatus from an ordinary intravenous bottle. The patient is in a prone position in bed. The zero level of the manometer is placed at the level of the xiphoid. The resting intrabiliary pressure is an index of the degree of obstruction to the flow of bile into the duodenum. The pressure normally runs from 0 to 30 mm. of water above the xiphoid level. If there is more pressure than this, the cause must be sought. It may be due to spasm, stricture, edema, pancreatitis, or stones. In the case of spasm the pressure is promptly reduced by inhalation of amyl nitrite.[3] Four deep inhalations should be taken by the patient immediately after the ampule is broken. In some cases, the spasm is superimposed on some organic cause, in which case the pressure will be only partially relieved by this method. Cholangiographic studies will determine whether or not there is obstruction from pancreatitis, stone, or stricture. Spasm may also be detected by this method. However, the degree of obstruction can be determined only by pressure studies. The cholangiogram in Fig. 1*B* indicates that there is obstruction at the lower

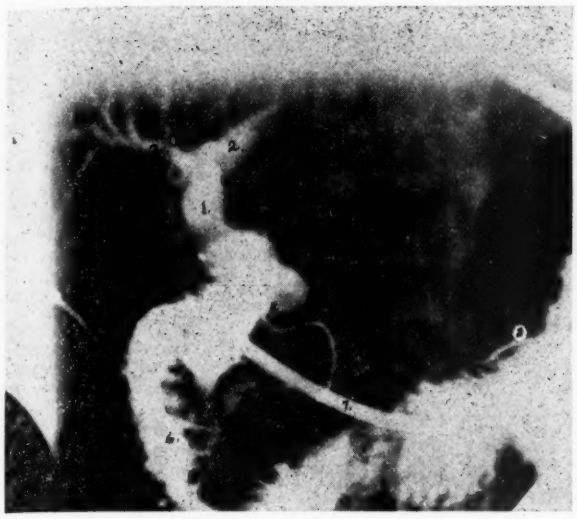
end of the common bile duct. Because this disappears in Fig. 1*C*, one may assume that this obstruction is from a spasm. Roentgenologically, this spasm appears to be of severe degree. However, the pressure studies show that this spasm represents only 50 mm. of water pressure. Since the secretion pressure of the liver is around 300 mm. of water, this degree of obstruction offers relatively little resistance to the flow of bile into the duodenum.

PERFUSION PAIN LEVEL

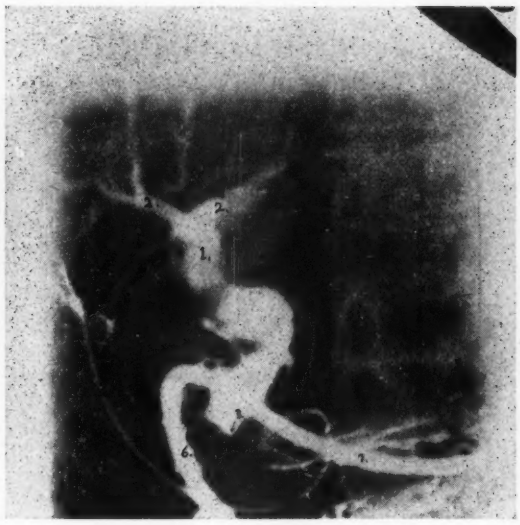
When the common bile duct is perfused with saline solution at increasing pressures, there comes a point when the patient experiences a sensation of discomfort. The pressure reading at this point is referred to as the perfusion pain level. This is measured in the following manner. A pressure apparatus is connected to the T tube. The system is filled with saline solution from an intravenous bottle. The intravenous bottle is then lowered until the level of solution in the bottle is the same as that in the manometer. The tubing connecting the bottle with the system is left open. The intravenous bottle is then gradually elevated. This results in gradually increasing pressure in the bile ducts and manometer. If no pain is experienced at 700 mm. water pressure, no further pressure is added. The first uncomfortable sensation which the patient feels, whether it be that of distention or actual pain, is used as an index of the pain level. The perfusion pain level is believed to be an index of the degree of inflammation within the biliary tract. It is very common to find a perfusion pain level of around 70 mm. of water three weeks after operation. With continued T-tube drainage the bile duct will become more resistant to pressure so that at the end of two to three months it will tolerate 500 to 700 mm. water pressure before pain is produced in the same individual. If the T tube should be removed when the pain level is low, one might expect that the patient would continue to have pain. If, on the other hand, the pain level is 500 mm. of water pressure, one would not expect that patient to suffer from postcholecystectomy pain because the secretion pressure of the liver is only 300 mm. of water, leaving a margin of 200 mm. water pressure before the pain level could be reached under normal circumstances.

After the pain level is determined, the patient is given ⅙ gr. morphine to test how much pressure will develop with duodenal spasm. This also serves as an index of the possibility of future postcholecystectomy pain. The amount of pressure produced by morphine spasm varies greatly between patients. An older woman, past the menopause, will frequently have a morphine pressure rise of no more than 50 mm. of water pressure. Such a patient would not be expected to suffer from biliary dyskinesia since this pressure would produce pain in only the very rare case of a patient with an extremely low pain level. On the other hand, if a morphine spasm produces a pressure rise of 300 mm. water as frequently occurs in young women, one would expect that patient to have attacks of postcholecystectomy pain. Such attacks can be relieved by inhalation of amyl nitrite or sublingual administration of nitroglycerin.

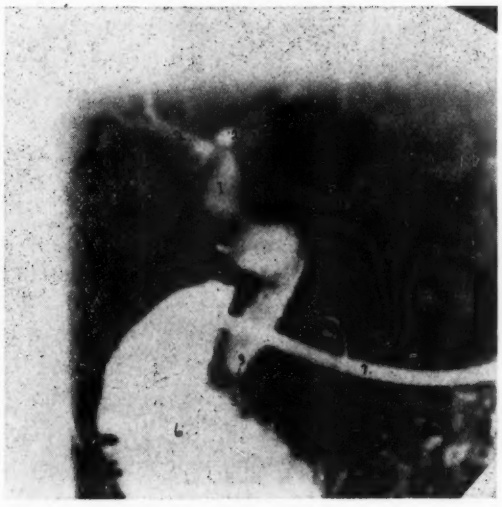
Fig. 1*A* shows a common duct with some narrowing at the lower end. This narrowing is not sufficient to produce any obstruction to the flow of bile. The resting pressure level in this instance was zero. Although roentgenologically there is some narrowing at the lower end of the bile duct due to a moderate degree of spasm of the duodenum, this is not sufficient to impede the flow of bile into the duodenum at a rate equal to that produced by the liver. The morphine response as indicated in Fig. 1*B* would lead one to believe that this



A.



B.



C.

Fig. 1.—*1*, Common bile duct; *2*, hepatic duct; *3*, point at which the lower end of the common bile duct begins to tunnel the musculature of the duodenal wall; *4*, pancreatic duct; *5*, ampulla of Vater; *6*, duodenum; *7*, duodenal tube passed through the nose; *8*, T tube in common duct.

A, This shows a moderate degree of spasm of the duodenum. The intramural portion of the common bile duct between *3* and *5* is definitely narrowed. The intramural portion of the pancreatic duct between *4* and *5* shows a narrowing for a shorter distance than that of the common bile duct since it tunnels the duodenum at a right angle rather than obliquely. The fact that the media regurgitates from the common bile duct to the pancreatic duct shows that there is some obstruction at the ampulla of Vater. This could be the nature of a fibrosis. A spastic duodenum would add to this narrowing since it would cause the folds of the duodenum to overlap each other. The muscle spasm of the duodenal wall, however, would not directly encourage regurgitation into the pancreatic duct of bile in this case since the pancreatic duct tunnels the duodenal wall independently of the common bile duct. Note also that there is no regurgitation of barium into the stomach. On the other hand, the barium is being carried at regular intervals downward into the jejunum, a condition which exists where there is not much spasm of the duodenum and probably not much irritability of the biliary tract from infection or otherwise.

B, This was taken ten minutes after subcutaneous administration of morphine, gr. 1/6. The duodenum is now in complete spasm. The lower end of the common bile duct is completely shut off as the result of this. There is regurgitation of barium through the pylorus into the stomach as indicated by the rugal markings above *7*. This indicates a relaxed pylorus. The condition in which a spasm of the second portion of the duodenum is associated with a reciprocal relaxation of the pylorus occurs very frequently. This is the beginning of the vomit reflex; it is associated with antiperistalsis of the duodenum.

C, This was taken two minutes after inhalation of amyl nitrate. Note the complete relaxation of the duodenum and the freedom with which the media runs from the common duct into the duodenum. Note that there is a slight regurgitation of media into the pancreatic duct indicating that there is some narrowing at the ampulla of Vater other than spasm, probably a fibrosis as a result of a previous duodenitis. Note that there is no reflux of barium into the stomach. The pyloric tone has recovered. Peristalsis now is in a downward direction toward the jejunum.

spasm was of a severe nature. However, pressure studies show that this only produces an obstruction sufficient to elevate the intrabiliary pressure to 50 mm. of water, well below the secretion pressure of the liver. This patient would not be expected to have any further trouble from biliary dyskinesia or the so-called postcholecystectomy syndrome particularly since the pain level is 500 mm. of water pressure. However, if the pain level were to be decreased markedly by cholangitis and the resting intrabiliary pressure increased by some organic condition such as inflammation of the head of the pancreas, one might expect the pain to occur in this case. Such a possibility is preventable by prolonged intrabiliary drainage, as will be later shown.

X-RAY OF THE BILIARY TRACT

X-ray of the biliary tract is done by injecting diodrast into the common bile duct through the T tube. The relation between the biliary tract and duodenum may be studied at the same time by injecting barium into the duodenum through an indwelling duodenal tube. In this way the relationship between the common bile duct, pancreatic duct, and duodenum can be studied. The diodrast is injected slowly until one feels a sensation of pressure against the plunger of the syringe, a total of 10 c.c. is usually enough. The injection is then stopped and the x-ray taken. At the same time, 60 c.c. of a thin solution of barium are injected through the duodenal tube into the duodenum. If the patient experiences pain at any time, the injection is stopped and the picture immediately taken. A series of three pictures are usually taken, one with the patient in the resting state, one following injection of morphine by ten minutes, and a third following deep inhalation of amyl nitrite by one minute. Cholangiographic studies will reveal the presence of stone, stricture, spasm, or pancreatitis.

Fig. 1*A* is a combined cholangiogram and duodenogram taken with the patient at rest. This shows a moderate degree of spasm of the duodenum indicated by a narrowing of this organ. The common bile duct enters the duodenum diagonally, showing a definite narrowing in the intramural portion of the common bile duct. There is considerable regurgitation of media into the pancreas. The pancreatic duct enters the duodenal wall in a different spot from that of the common bile duct. The intramural portion of the pancreatic duct is shorter since it enters the duodenum at a right angle. Since the two ducts tunnel the duodenal wall separately, one may assume that the regurgitation of media from the common bile duct into the pancreatic duct is due to some obstruction at the ampulla of Vater. This is due to a narrowing of the opening in the duodenal mucosa in this area.

It is believed that this stricture is the first lesion in the chain of events, resulting in regurgitation of bile into the pancreas producing pancreatitis. Following injection of morphine, in Fig. 1*B*, there is no regurgitation into the pancreatic duct. The reason for this is that the spasm of the duodenal wall has separated these two ducts. Fig. 1*C* shows a completely relaxed duodenum. The media runs freely from the common bile duct. Again there is some regurgitation into the pancreatic duct, indicating that spasm was not a factor in the production of this chain of pathologic disturbances.

I have made similar studies on several hundred cases over the past ten years. The results will be reported later. These studies indicate that one should leave the T tube in place until the following criteria have been satisfied.

1. The resting intrabiliary pressure must not be over 30 mm.
2. The perfusion pain pressure should be as high as 500 mm. water or better.

3. Roentgenograms of the common bile duct should show a free flow of media from the common bile duct into the duodenum with no evidence of stones or obstruction from edema or pancreatitis.

4. Two weeks following operation the T tube should be clamped at increasing periods of time starting with one-half hour twice daily and increasing by one hour per day. When the tube is being clamped off twenty-four hours of the day, one should wait another three weeks with a symptom-free patient before the tube is removed. At any time the patient has symptoms, the tube should be opened.

In this particular case there was a recurrence of pain of a biliary nature situated in the right upper quadrant of the abdomen, radiating around the subcostal margin and into the scapular regions ten days after the tube was clamped continuously. This attack was accompanied by chills and fever. There was prompt relief when the tube was opened. Continuous drainage was then instituted for another week. Gradual clamping was started again with permanent beneficial effects. After three weeks of having the tube clamped off all the time, it was removed. The patient has had no recurrence of symptoms since that time.

CASE REPORT

A 39-year-old white woman, a civilian dependent, was admitted to the Ashburn General Hospital, McKinney, Texas, May 28, 1944. Chief complaints were: (1) Pains in the right upper quadrant of the abdomen radiating into the scapular region, of one and one-half year's duration; (2) repeated attacks of diarrhea accompanied by blood and pus in the stool and repeated abscesses of the ischiorectal region for the past two years; and (3) intermittent chills and fever of one and one-half year's duration.

Past History.—The patient had typhoid fever in early childhood. At the age of 13 and again at the age of 18, she had malaria. In August, 1943, she had an attack of malaria; parasites were found in the blood. There was a history of chills and fever in 1939, diagnosed as pyelitis. In the years 1942 and 1943 she had repeated ischiorectal abscesses which were incised and drained. She was left with a persistent fistula-in-ano which was excised April 20, 1944, in a civilian hospital. The wound from this operation continued to drain until her admission to this hospital.

History of Illness at Time of Admission.—One and one-half years before admission the patient began to suffer from sharp pains in the right upper quadrant of the abdomen, fifteen minutes after meals. The patient usually vomited, with complete relief. Pain radiated through to the right scapular region. Because of the inability to retain food, the patient's weight decreased from 180 to 130 pounds.

Physical Examination.—On admission the abdomen was flat. There was some tenderness in the epigastrium and in the right upper quadrant over the gall bladder region. The patient was in such a poor state of nutrition at the time of admission that it was deemed inadvisable to give her a dye in order to x-ray the gall bladder. A flat plate of the abdomen taken at that time was negative. There were healing wounds in the region of the anus, the site of previous operations for fistula-in-ano.

Progress in Hospital.—The patient was put on bed rest and bland diet on admission. She improved considerably. The discharge of blood and pus from the rectum continued. At 4:00 A.M., June 7, 1944, the patient developed a sudden severe pain in the right upper quadrant of the abdomen. This was more severe than it had been on any previous occasion. It kept her awake the remainder of the night. She vomited considerably. At 9 A.M. the white blood count was 15,000. Physical examination at that time revealed considerable tenderness over the gall bladder with a moderate degree of spasm of the muscles in this region. At 3 P.M. of the same day the pains became even worse and were more diffuse over the abdomen. The white blood count rose to 25,000. Physical examination at that time revealed a great deal of spasm and tenderness over the upper part of the abdomen, with tenderness and rebound tenderness extending even to the left lower quadrant. Because of the severity of the symptoms, it was decided that immediate operation was necessary in spite of the poor state of the patient's nutrition. The operation was done, using local anesthesia supplemented by gas oxygen and ether inhalation anesthesia.

Operative Findings.—The gall bladder was markedly distended to three times its normal size. The walls of the gall bladder were very thickened and edematous. There was a considerable amount of purulent plastic exudate around the gall bladder, the gastrohepatic omentum, and common duct. There was a great deal of free semipurulent fluid in the peritoneal cavity. The common duct was enlarged to the size of a thumb. The pancreas was greatly inflamed and thickened. This inflammation spread over the head and all of the body of the pancreas. It was approximately 6 cm. in thickness in its anteroposterior diameter.

Operative Procedure.—A right upper quadrant midrectus incision was used. The gall bladder was removed from below upward. It shelled out readily from its bed. The common duct was opened and explored; no stones were found. A T tube was placed in the common duct for continued prolonged biliary drainage. The gall bladder did not contain stones.

Postoperative Convalescence.—The patient had an uneventful postoperative convalescence. Two weeks postoperatively routine pressure studies were made on the bile duct, and cholangiograms were taken. The resting intrabiliary pressure was 0. The pressure following injection of morphine was 50 mm. of water, the perfusion pain pressure was 500 mm. of water. Roentgenographic examination taken at rest showed the duodenum to be in a moderate state of spasm. There was some narrowing of the lower end of the common bile duct and there was some regurgitation of bile into the pancreatic duct (Fig. 1*A*). Following injection of morphine there was a great deal of duodenal spasm (Fig. 1*B*). Following inhalation of amyl nitrite, the duodenal spasm was completely relaxed and the media flowed freely from the common duct into the duodenum (Fig. 1*C*). There was still some regurgitation of bile into the pancreatic duct indicating a small degree of stricture at the ampulla of Vater.

Clamping of the T tube was then begun, beginning with one-half hour twice a day and increasing by one-half hour twice a day. After the tube was clamped off continuously night and day for a period of ten days, the patient developed chills and fever. She also complained of a pain in the right upper quadrant of the abdomen radiating into the right scapular region. The tube was then opened and drainage re-established. These symptoms promptly cleared up. After drainage for another week, the clamping began as before. This time the clamping continued until the tube was completely clamped off for a period of three weeks, and the patient was entirely symptom free. The T tube was then removed. The patient has been symptom free since that time so far as the biliary tract is concerned.

Postoperatively, pus and blood continued to be discharged from the rectum. Proctoscopic examination revealed the presence of ulcerative colitis, moderately severe. Roentgenologic examination of the colon confirmed these findings. She was then given sulfasuxidine for one week followed by sulfathiazole for another week with excellent results. The discharge cleared up. The number of stools were reduced from ten a day to one a day.

DISCUSSION

The chain of events in this case occurred probably as follows: The narrowing of the outlet of the ampulla of Vater resulted in regurgitation of bile from the common bile duct into the pancreatic duct. Pancreatitis developed. The swelling in the head of the pancreas produced obstruction of the common duct, resulting in cholangitis and cholecystitis. When the gall bladder contracted in an attempt to empty the bile into the duodenum, the regurgitation of bile into the pancreatic duct increased. This aggravated the pancreatitis. Cholangitis and cholecystitis were made worse, and so a vicious cycle resulted. One would expect removal of the gall bladder to reduce the potential intraductal pressure to that of the secretion pressure of the liver and so tend to cut down the regurgitation of bile into the pancreas. In such case, therefore, cholecystectomy would be expected to be beneficial in clearing up the pancreatitis.

The patient had periodic attacks of chills and fever. Since she lived in a malaria area, this syndrome constituted a diagnostic problem. It is very probable that several of the alleged attacks of malaria were actually due to cholangitis. Cholangitis should always be considered as a cause of chills and fever. The cholecystectomy in this type of case must be looked upon as only one step in the correction of the physiologicopathologic disturbances present. Continued and prolonged drainage of the common duct must be considered of great importance.

In general, in the postoperative care of patients with diseases of the gall bladder, the use of opiates should be avoided since they produce marked duodenal spasm and back pressure in the common duct. However, in a patient with T-tube drainage, duodenal spasm can do no great harm since there is an escape of bile through the T tube. It has been found that dilaudid is a much more desirable analgesic in these cases since it produces less duodenal spasm. Occasionally even dilaudid produces nausea and vomiting from duodenal spasm. This effect can be neutralized by the addition of nitroglycerin, $\frac{1}{100}$ gr. to each dose given. In using nitroglycerin to relax duodenal spasm, one must be sure to obtain a brand that has a consistent potency. Some of the brands studied, particularly the tablet triturates, were found to be relatively inert. The hypodermic tablets are more consistent in their potency. The nitroglycerin granules are more desirable for sublingual administration.

In the case reported, the spasm was not of a prime factor, since the common bile duct and pancreatic duct entered the duodenal wall through different tunnels. If the pancreatic duct and common bile duct had joined before entering the duodenum, as occurs in some cases, the use of morphine would be very detrimental. In such cases the spasm would produce an obstruction beyond the union of the two ducts and so result in more regurgitation of bile into the pancreas. The roentgenogram shown in Fig. 1*B* indicates that the duodenal spasm acts separately on the two ducts and that there is no regurgitation into the pancreatic duct in spite of marked duodenal spasm.

Just what relationship the chronic ulcerative colitis had to the pancreatitis and cholangitis is difficult to say. It is very probable that the disturbance of nutrition as a result of malfunction of the pancreas and liver resulted in lower tissue resistance and was a factor, at least, in the persistence of the ulcerative colitis. With this in mind, a great deal of attention was paid to the patient's nutrition. She was given liver extract intramuscularly and was given all the vitamins as well. The reason for giving sulfasuxidine and sulfathiazole alternately is as follows: The sulfasuxidine acts within the bowel lumen, reducing the number of bacteria present. However, it has no beneficial effects at the base of the ulcers or in the lymphatics in the bowel wall. The sulfathiazole takes care of the latter.

SUMMARY

1. A case is presented in which it is believed that stricture of the ampulla of Vater produced a regurgitation of bile into the pancreatic duct and pancreatitis. Pancreatitis then produced obstruction to the common bile duct and gall bladder resulting in cholangitis and cholecystitis.

2. Chills and fever, common symptoms of cholangitis, may be confused with malaria in malaria areas.

3. Removal of the gall bladder is only one step in the right direction in the treatment of chronic pancreatitis and cholangitis. It should be accompanied by T-tube drainage of the common bile duct. The T tube should not be removed until all evidence of pancreatitis and cholangitis have disappeared.

4. Spasm of the duodenum, an important factor in postcholecystectomy syndrome, is of no importance in this particular case since the common bile duct and pancreatic duct enter the duodenal wall through separate tunnels opening into the ampulla of Vater inside the duodenum.

5. The T tube must not be removed too early. It should not be removed until: (a) The resting intrabiliary pressure is 30 mm. or less of water; (b)

the perfusion pain level is 500 mm. or more; (c) roentgenologic studies of the common bile duct show the absence of any obstruction to the flow of bile into the duodenum. Finally, the T tube should be continuously clamped for a period of three weeks with the patient entirely symptom free.

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SPONTANEOUS RUPTURE OF LOWER COLON WITH EVISCERATION OF SMALL INTESTINE THROUGH THE ANAL ORIFICE

A Complication of Advanced Rectal Prolapse

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THE protrusion of loops of small intestine through the anal orifice is a bizarre sight which has only occasionally been observed. It depends upon certain etiologic factors and pathologic states which are interesting to consider. A survey of the literature reveals only six previously recorded cases. Our case, reported herewith, has stirred us to seek a common explanation for this rare type of evisceration in these several patients. A review of case histories brings to light the fact that in six spontaneous cases, including ours, the patients were known to be previously afflicted with an advanced state of rectal prolapse (procidentia); the seventh, and remaining case, that of a young girl, was caused by severe crushing trauma without antecedent prolapse. Consequently, it can be stated that rectal prolapse is an etiologic factor of prime importance in the occurrence of this rare type of evisceration. And conversely, one can classify spontaneous rupture of the lower colon and evisceration through the anal orifice as a definite complication, though an unusual one, of advanced rectal prolapse.

CASE REPORT

J. T. A., a 76-year-old unmarried woman, was admitted to the Union Memorial Hospital, the evening of Oct. 15, 1942. Since June, 1933, she had been a patient at the Home for Incurables, afflicted with advanced chronic arthritis. She had been known to suffer from hemorrhoids and had been examined by one of us (S. McL.) several years previously in connection with an extensive rectal prolapse, which had been the cause of considerable distress. However, she was able to be up and about a part of the time and could feed and care for herself. The following description of what took place late in the afternoon of Oct. 15, 1942, was supplied by the nurse who attended her. "In an attempt to get from the commode to the bed, the patient fell to the floor. She was found lying on the floor with what seemed to be the lower intestines hanging from the rectum and a moderate amount of blood surrounding same. I was able to observe persistalsis of the intestines. Intestines were then covered with sterile linen and the patient lifted into bed." The medical director of the Home saw her

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immediately and requested surgical help. Fig. 1, drawn from a colored photograph taken at the time, illustrates the general appearance of the evisceration. The patient was transferred at once to the Union Memorial Hospital.

Upon examination, temperature was 98° F., pulse 84 beats per minute, and respiratory rate 20. She looked her age, and was somewhat senile mentally. She did not appear to be in much pain, nor did she present the usual characteristics of shock. There was a marked dorsal kyphosis with ankylosis of the spine and an ankylosis of both hips in a position of 35 degrees of flexion. The heart and lungs presented no gross abnormality. The abdomen was not distended and its walls were moderately well relaxed. The most remarkable finding was the presence of approximately five feet (152 cm.) of small intestine lying between the patient's thighs, the loops having eviscerated by way of the anal orifice. The serosal surfaces were apparent. The color was good and normal peristalsis was taking place. There was no protruding rectal mucous membrane. Examination of the anal canal showed the sphincter to be very relaxed. The afferent and efferent loops were definitely traversing the anal canal and the site of their egress from the peritoneal cavity could not be determined.

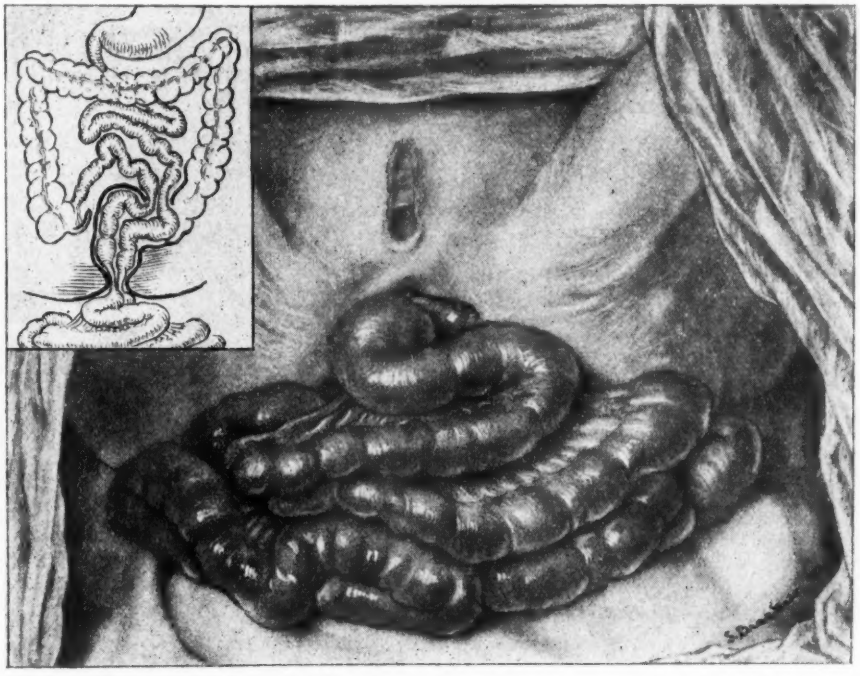

Fig. 1.—Drawing taken from colored photograph showing evisceration of small intestine through anal orifice following rupture of the lower sigmoid in a case of recurrent rectal procidentia.

A future attempt was made to manipulate gently the intestinal coils in order to replace them. Laparotomy was decided upon and under ether anesthesia a low midline incision was made (M. L. J.). When the peritoneal cavity was opened, the explanation of the trouble was at once apparent. There was a rent in the anterior wall of the sigmoid colon, about six inches (15 cm.) above the peritoneal reflection on the rectum. This rent was about 2½ inches (6 cm.) long and did not appear to represent a pathologic area in the colon. Through it was protruding a large part of the small intestine, much of which in turn passed through the anal orifice to the exterior. The intestine was quickly drawn back into the peritoneal cavity, care being taken to sponge off any visible foreign particles. It was found that the loop of sigmoid which had been torn would reach to the anterior abdominal wall and consequently it was brought out through a left muscle-splitting incision as a colostomy. This was done with the idea of avoiding a repetition of the accident which had just occurred. Crystalline sulfanilamide in the amount of 7 Gm. was placed in the peritoneal cavity, 3 Gm. being used for the wound margins. The wound was quickly closed with buried chromic catgut and silk in the skin, and the patient left the operating room in fair condition.

There was no significant elevation of temperature or pulse during the postoperative period, nor was there vomiting or distention. On the day following operation, the leucocytes

numbered 6,600 and the hemoglobin was 76 per cent. Despite intramuscular administration of prostigmine and pitressin, no bowel movement occurred. Quietly but quickly she died, about sixty-two hours after operation.

Post-mortem examination disclosed a widespread confluent pneumonia, and on the right side an esophagopulmonary fistula, the walls of which showed evidences of acute inflammation. Within the abdomen there were many fresh adhesions and several small abscesses, indicating peritonitis. The stomach and duodenum were moderately distended. There was marked arteriosclerosis.

The following are abstracts of six previously reported cases.

CASE 1 (Moulton[1]).—The patient was an insane man who had acquired the ability to produce rectal prolapse and inversion at will. One day the attendant discovered him with a loop of small intestine two feet in length hanging with its mesentery from the rectum. There was a tear in the wall of the rectum through which the small intestine protruded. The patient was said to have died in thirty-six hours "from shock." There was no operation.

CASE 2 (Guibe[2]).—A 78-year-old woman gave a long history of rectal prolapse. Having an imperative desire to go to stool one day and making a violent effort, she experienced a sharp pain and a mass of small intestine escaped through the anal orifice. The following morning, a laparotomy was performed under local anesthesia, and it was necessary to resect 130 cm. of gangrenous, protruding bowel. There was a tear, 3 cm. in length through the anterior band of the sigmoid, about 10 cm. above the cul-de-sac. This was closed. Eight days later, a fecal fistula developed. After six weeks she left the hospital with a small fistula, but became cachectic and died one month later.

CASE 3 (Guide[2]).—A 77-year-old woman with a history of prolapse of the rectum fell down the stairs, sustaining a painful contusion in the region of the coccyx and experiencing a sensation of something breaking. Upon examination it was ascertained that 1.5 meters of small intestine and 30 cm. of sigmoid and descending colon were protruding from the anus. The rupture was situated at the intraperitoneal part of the rectum about 3 cm. above the base of the cul-de-sac. The rent was 9 cm. long and the bowel was otherwise in good condition. The rectum was sutured, peritoneal cavity irrigated, and sigmoidostomy performed. She died on the fifth day with signs of peritonitis. Autopsy showed that "the loops of intestine for a distance of 2 meters terminating at the ileocecal valve were dark red and there were many suffusions of blood in the intestine and mesentery."

CASE 4 (Moskalenko[3]).—This patient was a 52-year-old woman with a fifteen-year history of rectal prolapse. On the morning of admission she had defecated, apparently out in the fields, when seized with a sudden severe cough. She experienced an immediate, sharp abdominal pain during which her intestines were extruded through the rectum. When first seen, the intestines were cyanotic and covered with earth and straw. Her general condition was fair. Laparotomy was performed fifteen hours after the accident. Fecal soiling of the peritoneum was present. About one-half of the small bowel had prolapsed outside and resection of this amount was necessary. The tear in the rectum, 8 cm. in length on the anterior surface near the bottom of the pouch of Douglas, was sutured. The patient died in twenty-four hours of heart failure. At autopsy attention was directed chiefly to the cul-de-sac of Douglas. It was found that this pouch was much deeper and fuller than normal and that when pressure was applied, only a thin layer of bowel wall intervened to prevent extrusion of the abdominal viscera.

CASE 5 (Erlikhmana[4]).—A 45-year-old laundress with a history of rectal prolapse was stricken with a severe pain in the lower abdomen while carrying a heavy load of wet laundry. There was an evisceration through the rectum of almost the entire small intestine. Laparotomy was performed under ether-chloroform anesthesia; the intestines were pulled back into the peritoneal cavity and washed with normal salt solution. The rectal wound, which was on the anterior wall of the pelvic portion of the rectum, was sutured in two layers. The patient died in sixty-four hours with generalized peritonitis. Examination of the ruptured area of the rectum revealed an ulcer measuring approximately 9 by 1.5 cm., running transversely to the long axis of the gut. The microscopic examination of the ulcerated portion of the rectum revealed an ordinary scarred ulcer having no specific characteristics.

CASE 6 (Zena, D.[5]).—(This was not a spontaneous rupture but one which resulted from external violence.) A 12-year-old girl suffered multiple injuries when caught in a cave-in. Approximately 80 cm. of small intestine were found to protrude from the anus. At laparotomy,

the intestine was replaced and the injured area of the rectum excised and sutured. A drain was introduced. The patient was out of danger in two days. Anal incontinence and some diarrhea were present for a short time, but complete recovery eventually occurred.

DISCUSSION

Manifestly, in an otherwise normal individual, the complication to be expected to accompany a rupture or laceration of the intestine is the free passage of intestinal contents into the peritoneal cavity. In this less common type of rupture of the intestine, the prime complication is the passage of peritoneal cavity contents into the lumen of the bowel and, indeed, on to the outside. Both Lynch[6] and Bacon[7] in their textbooks have mentioned rupture of the protruding rectal wall with evisceration as a serious but infrequent occurrence in rectal prolapse. The pathologic anatomy which permits intra-abdominal forces to tear the lower intestine in this fashion is, of course, the root of the trouble.

In advanced rectal prolapse or in procidentia, a single thickness of the wall of the upper rectum or sigmoid is called upon to assume all of the intra-abdominal stress without the aid of the normally supporting structures. Among these structures are the intact pelvic floor and the competent anal sphincter. In women, the area of rectal wall subject to the greatest stress is that which lies anteriorly in Douglas' cul-de-sac. The very fact that its serosa is reflected anteriorly and finally becomes fixed contributes in no small measure to the susceptibility of this bowel segment when the proper forces are in effect. With the rectum in a prolapsed position and with the hernia-like pouch formed by the anterior wall of the rectum and sigmoid filled with small intestine, it is not difficult to appreciate how rupture and sudden evisceration may occur.

Wangensteen[8] quotes the work of Hay (1940) in determining the breaking strength of the human bowel removed at operation. In a series of ten patients the bursting strength was noted to vary between 80 and 260 mm. of mercury, truly a wide range. This type of experiment, however, represents a different approach to the problem as it deals with intraluminal pressures and is not entirely applicable to external (intraperitoneal) pressures and stresses.

There is little to advance or suggest with regard to treatment. The patients must be managed according to accepted principles for abdominal trauma, which will necessarily mean laparotomy with repair of the defect. Resection has been carried out several times. It is a situation requiring judgment and initiative adapted to each case. The results in the reported cases have been poor. Of the six spontaneous ruptures, five were operated upon and only one patient survived for one month. The young girl who had a traumatic rupture with evisceration unaccompanied by an antecedent prolapse recovered. It is true that advanced age and unfavorable physical condition often accompanied the immediate catastrophe. Consequently, it may be concluded, as in other types of large intestinal wounds and perforations, that the prognosis is not good. This very fact of poor prognosis, however, presents an added reason for the early surgical correction of rectal prolapse before it reaches an advanced and complicated state.

CONCLUSIONS

1. A case of spontaneous evisceration of small intestine through the anal orifice following a rupture of the sigmoid colon is reported.

2. A review of six previously reported similar cases reveals the interesting fact that in five of these as well as in our own, the patients had had a pre-existing rectal prolapse.

3. This observation leads to the conclusion that rectal prolapse (procidentia) is an etiologic situation of prime importance in the occurrence of this rare type of evisceration.

4. The pathologic anatomy permits the requisite force to be directed against the anterior wall of the rectum or colon in a manner to cause rupture when procidentia exists and when that wall is no longer supported by the intact pelvic floor and the competent anal sphincter.

5. The poor prognosis in this late complication presents an added reason for the early correction of rectal prolapse.

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THREE HUNDRED FIFTY-TWO CASES OF TETANUS

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THERE were 352 patients with tetanus treated in the Charity Hospital of Louisiana at New Orleans during the ten-year period of Nov. 1, 1934, to Oct. 31, 1944. The total mortality was 45 per cent. There has been previously published in the medical literature four articles which together with this one, review the treatment given all patients with tetanus treated at Charity Hospital during the years of 1906 to 1944. In 1935, Boyce and McFetridge[1] reported 185 cases, with a mortality of 59.5 per cent for the period from Jan. 1, 1930, to Oct. 31, 1934. In 1930, Graves[2] reported 217 cases, with a mortality of 52 per cent for the period from 1923 through 1929. In 1916, Gessner and Adiger[3] reported 368 cases covering the period from 1906, with a mortality of approximately 70 per cent, and in 1924, Graffagnino and Davidson[4] brought this number of cases to 596, with a mortality for the combined series of 67.4 per cent.

Vener and Bower,[5] in 1941, reported 100 cases during an unstated period from the Los Angeles County General Hospital, with a mortality of 29 per cent. They, together with McKillop,[6] had reported, in 1933, a series of 131 cases, with a mortality of 50 per cent. Moore and Singleton,[7] in 1939, reported 102 cases from 1906 to 1939 at the John Sealy Hospital in Galveston, Texas, with a mortality of 50 per cent. Kirtley[8] of Nashville, Tenn., reported 60 cases from 1925 to 1938, with a mortality of 46.6 per cent. In 1940, Dietrich[9] reported 28 cases from the Children's Hospital in Hollywood, Calif., with a mor-

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tality of 47 per cent. He emphasized the fact that from 1921 to 1932, 12 of 15 of these children died, while from 1932 to 1939 only 1 of 13 children died. He attributed most, if not all, of these deaths to faulty methods of administration and to too large doses of the tetanus antiserum.

The yearly distribution and mortality of this series are shown in Table I.

TABLE I

NUMBER OF CASES	YEAR	NUMBER OF DEATHS	PER CENT
5	Nov. & Dec., 1934	3	60
41	1935	21	51
32	1936	20	51
33	1937	13	40
41	1938	24	58
24	1939	11	46
43	1940	21	49
42	1941	17	40
26	1942	8	30
31	1943	11	35
34	1st 10 mo., 1944	9	26
Total 352		158	45

One of the main purposes of this article is to emphasize the markedly lowered mortality of 31 per cent among the 91 consecutive cases of the last three years and to outline the therapeutic program which, it is believed, will further lower the tetanus mortality rate.

The monthly distribution and mortality are given in Table II. It is interesting to note that in general the summer months show a higher incidence of the disease but have a much lower mortality.

TABLE II

MONTH	NUMBER OF CASES	DEATHS	PER CENT
Jan.	21	14	67
Feb.	14	7	50
March	27	16	59
April	33	20	61
May	42	14	33
June	39	12	31
July	33	11	33
Aug.	27	11	41
Sept.	46	16	35
Oct.	36	19	53
Nov.	19	10	53
Dec.	15	8	53

The ratio and comparative mortality of age, race, and sex are listed in Table III.

Vener and Bower[5] reported a mortality in their series of 52 per cent among 25 females and a mortality of 21.3 per cent among 75 males. It is ap-

TABLE III

RACE, SEX, AGE	NUMBER OF CASES	DEATHS	PER CENT
Negro	217	103	47
White	135	55	41
Male	229	106	46
Female	123	52	42
Adults (over 12 yr.)	189	91	48
Children	163	67	41

parent from this tabulation that no such higher female mortality existed in this series, which in fact shows a surprisingly uniform rate of mortality for age, race, and sex.

DIAGNOSIS

In the diagnosis of tetanus the familiar pattern of recent injury, trismus, dysphagia, stiff neck, generalized rigidity, and convulsions probably provides at least as consistent a symptomatology as does any communicable disease. Needless to say, there are variations and atypical clinical pictures. It is certain, however, that if tetanus were seriously considered in the differential diagnosis of any patient presenting two or more of these symptoms, very few cases of tetanus would be misdiagnosed.

In this series there were 26 cases which apparently presented certain definite symptoms or signs of tetanus, but which were unrecognized by the physician at the time of the first examination. Twenty-two of these patients were seen in the emergency or admitting rooms of Charity Hospital by interns or residents, and 4 were seen by private physicians.

Of the 4 patients who were seen by private physicians, 1 who had complaints of trismus and generalized rigidity was diagnosed as having worms and sent home; 1 patient who was having convulsions had no diagnosis and was sent home, as were the other 2 patients who had speech impairment, trismus, and some muscular rigidity.

Eight of the patients who were seen at Charity Hospital were sent home; 3 of them gave a history of having convulsions, and the other 5 had two or more of the symptoms of trismus, stiff neck, muscular rigidity, or contractions.

Certainly the criticism, if any, of the handling of these cases should be directed not toward the failure in diagnosing them correctly, but rather the failure to hospitalize and study any patient presenting such symptoms. Dietrich[9] stated that of the 28 cases in children reported in 1940, only 17 were diagnosed as tetanus at the time of hospital admission.

Fourteen patients were admitted to the hospital and treated for variable lengths of time (one to seven days) under various diagnoses before the diagnosis of tetanus was made and antitetanus treatment instituted. The tentative diagnoses on these patients were as follows: deferred, 3; hysteria, 3; meningitis, 3; carcinoma of esophagus, 1; chronic otitis media and mastoiditis, 1; pneumonia, 1; cellulitis of face, 1; undiagnosed autopsy, 1. The mortality for this group of patients was 14 deaths, or 56 per cent. There were also 2 cases tentatively diagnosed as tetanus until they came to autopsy. One of these patients was found to have a generalized peritonitis and toxemia from a traumatic perforation of the right colon. The other had pulmonary tuberculosis and a brain tumor.

PROPHYLAXIS

It is estimated that an average of 65 patients daily has received antitetanus serum at Charity Hospital for the last ten years. This would give a total of 237,250 patients receiving the serum during this period of time.

Ten patients who had received a prophylactic dose of antitetanus serum at the Charity Hospital emergency rooms within twenty-four hours of the injury, later developed tetanus. Six of these patients died, a mortality of 60 per cent. Nine of these patients had received 1,500 units of antitetanus serum. The remaining patient received a double dose of mixed antitetanus and gas gangrene serum.

Assuming that all patients who received antitetanus serum at Charity Hospital return there for treatment should tetanus develop, we find a mortality

for tetanus of 24 per million of those who receive the prophylactic antitoxin. This compares favorably with the records of the Bureau of Vital Statistics, which give an annual mortality in the state of Louisiana of about 40 per million of population.

The records of the Bureau of Vital Statistics[10] reveal that tetanus is most prevalent in the states immediately surrounding the Gulf of Mexico. The incidence steadily diminishes as one progresses northward. It is rare in the far northeastern states and in the states west of the western borders of Texas, Kansas, Nebraska, and Minnesota, except for a moderate incidence in Arizona and California.

The list of injuries from which tetanus has developed in this series includes many insignificant scratches, minor abrasions, or minute puncture wounds. When one considers how many of such types of injuries are received each year by the average person it is clearly illogical to expect or to suggest that antitetanus serum be given for each such injury. However, from studying the types of wounds from which most of these cases of tetanus have developed, there emerge certain very definite indications in the tetanus-prevalent areas for débridement and prophylactic antiserum. Lay people should be educated to recognize these indications and to seek medical care for each such type of injury. Foremost among these are puncture wounds, second are splinters, and third are all secondarily infected and suppurating wounds. To these may well be added burns, subdermal lacerations, and infected surgical incisions. It should also be remembered that the duration of the passive immunity produced by the antitoxin is not more than eight or ten days, and should therefore be repeated at such intervals for wounds that remain open or chronically infected for longer periods of time. It is also believed that especially in the more tetanus-prevalent areas no physician can safely treat any traumatic lesion, however insignificant, without also administering antitetanus serum.

For minor traumatic lesions which will not receive a physician's care, probably the next best insurance against tetanus is an immediate thorough scrubbing of the wound with soap and water. This treatment is, of course, entirely ineffective in conditions previously described in which débridement and antitoxin are definitely indicated.

It is hoped that the time will come soon when routine immunization by tetanus toxoid among the population of tetanus-prevalent areas will be done. Certainly the efficacy of such a procedure has been proved by the experience of the Armed Services in World War II, and at present appears to be the only known solution to the prevention of the disease. The United States Army method of toxoid administration[11] is to give three injections of 1 c.c. each at monthly intervals, followed by a booster dose of 1 c.c. at the end of twelve months, and thereafter an additional 1 c.c. in the event of an injury, providing more than six months have elapsed since the previous injection.

CLASSIFICATION OF CASES

For a more detailed study the cases have been classified according to origin of infection as follows: tetanus neonatorum, gynecologic, surgical, chronic skin lesions, unknown, and traumatic. These will be considered separately.

Tetanus Neonatorum (11 Cases).—There were 11 patients with tetanus neonatorum in the series, all of whom showed evidence of umbilical cord infection. Six of these patients died, a mortality of 55 per cent. One of these patients died on the first hospital day, 2 died on the second, 2 on the third, and 1 on the ninth hospital day. Of the 5 who recovered, their period of hospitalization varied

from twenty-one to forty-five days, an average of thirty-three days. The age of the patient at the time of onset of symptoms varied from 8 to 12 days.

Three were unattended deliveries, 2 at home where no dressing was placed over the umbilicus of 1, and 1 was born in an automobile; 2 others were also home deliveries, 1 attended by a private physician and 1 attended by a medical student. The circumstances of birth of the other 6 are not recorded. All these patients were hospitalized and antitetanus treatment instituted within from two to four days of onset of symptoms. Six of them received 20,000 to 25,000 units of antitetanus serum intramuscularly on admission. Three of these died on or before the third day and the other three recovered. One patient received 20,000 units intravenously on admission, but received no subsequent doses and died on the third day. One patient received 60,000 units intravenously and 20,000 units intramuscularly on admission, with no subsequent doses, and died on the ninth day. Three patients received 40,000 or 50,000 units intramuscularly on admission, 2 of whom recovered, and the other 1 died on the third day. Eight of the 11 patients received no débridement, of whom 5 died. Umbilectomy was done on 3 patients of whom 1 died.

It would seem well at this point to re-emphasize the fundamental principle of giving antitoxin to children, namely that the antitoxin is to be given in sufficient quantities to neutralize the toxin. A focus of infection will produce as much toxin in an infant as it will in an adult. Hence, no reduction in the amount of antitoxin for a child or infant, however small, is permissible. The inescapable conclusion is that the mortality for this particular group of patients could have been appreciably reduced by routine umbilectomy and initial doses of 80 to 100,000 units of antiserum by the intramuscular and intravenous routes.

Surgical (11 Cases).—Eleven of the cases followed a surgical procedure. Six of these patients died, a mortality of 55 per cent. The periods of incubation varied from two to twenty days, an average of nine days. Five of these cases followed extraction of teeth, from which 3 patients died. One case followed a combined appendectomy and tonsillectomy, 1 a hernioplasty with resection of bowel, 1 a hysterectomy, 1 a ganglionectomy, and 1 an excision of a cheek lesion. The incisions of all these patients showed evidence of gross infection from one to four days before the signs of tetanus developed. The remaining case followed a hypodermic injection by a private physician to a school child.

Chronic Skin Lesions (9 Cases).—There were 9 cases where the site of infection was thought to be in chronic skin lesions which had been present for at least several months. Six of these patients died, a mortality of 67 per cent. Four of these patients had chronic leg ulcers, 1 had an infected skin rash, 1 a chronic sore on the penis, 1 multiple decubitus ulcers, 1 a chronic draining osteomyelitis, and 1 an infected draining ear.

Gynecologic (14 Cases).—There were 14 patients with gynecologic tetanus, 6 of whom died, a mortality of 43 per cent. Twelve of these followed abortions, 8 of which were induced by midwife manipulations, 1 was self-induced with a catheter, and 3 were recorded as of unknown cause. One case developed following an uncomplicated hospital delivery, and the remaining patient developed tetanus following an induced labor on an eclamptic by means of a Voorhees bag.

Unknown (38 Cases).—In 38 of the cases no history of any injury could be elicited and no possible site of infection found. Twelve of these patients died, a mortality of 32 per cent.

TABLE IV

LOCATION	NUMBER OF CASES	AVERAGE INCUBATION PERIOD (DAYS)	DEATHS	PER CENT
Lower extremity	205	12.6	91	44
Upper extremity	42	10.2	24	57
Head	17	8.6	9	53
Trunk	2	10.5	0	--
Multiple	3	----	1	33

Traumatic (269 Cases).—Two hundred sixty-nine cases in the series gave a definite history of recent injury or presented a traumatic site of infection. The incubation period varied from 1 to 100 days, an average of twelve days. The anatomic distribution was as shown in Table IV.

The distribution of the types of wounds is listed in Table V.

TABLE V

WOUND	NUMBER OF CASES	AVERAGE INCUBATION PERIOD (DAYS)	DEATHS	PER CENT
Puncture	101	12.7	50	50
Splinter	69	10.2	30	43
Laceration	54	11.9	26	46
Scratch	11	7.2	5	45
Abrasion	16	9.0	6	38
Burn	3	20.0	1	33
Bite (1 dog, 1 rat)	2	30.0	1	50
Compound fracture	3	11.7	3	100
Crushing	3	14.7	1	33
Amputation of finger	2	10.0	0	--
Multiple	4	----	1	25
Abscess	1	5.0	0	--

Of the 101 puncture wounds, 71 were from nails, 7 unknown, 5 tacks, 5 bone, 3 pins, 2 wires, 2 pitchforks, 2 rakes, 1 ice pick, 1 needle, 1 shoe nail, 1 steel tong. Of the 69 splinters, 58 were wood, 3 thorns, 2 steel, 1 soybean barb, 1 weed, 1 bone, 2 shells, and 1 unknown. Of 54 lacerations, 18 were caused by unknown objects, 16 by glass, 4 brick, 3 tin, 2 shell, 2 cement walks, 2 knives, and 7 miscellaneous. The ages of the patients in this group ranged from a few months to 74 years. It is very evident that no age or class of people is immune and that no injury is too trivial to cause tetanus.

In an attempt to evaluate the relationship between the length of the incubation period and the mortality rate, 253 patients were selected who gave a definite history of injury with a known incubation period (see Table VI).

TABLE VI

INCUBATION PERIOD (DAYS)	NUMBER OF PATIENTS	DEATHS	PER CENT
1-5	43	19	44
6-10	97	60	62
11-15	60	22	37
16-20	24	7	29
20-50	26	7	27
51-100	3	0	--

These statistics agree with the generally accepted view that longer incubation periods are associated with lower mortality rates. The presence of a lesser mortality rate among the patients in the one- to five-day incubation

period group than in the six- to ten-day group is in decided contrast to most other published series of cases. However, it should be remembered that all cases with a history of a very short or a very long incubation period should be regarded with some doubt, as the true cause of infection may have occurred unnoticed or forgotten at some other time.

TREATMENT

The experimental work of Firor[12] indicates that the tetanus toxin is carried from the focus of infection by the lymphatics and blood vessels. In the tissue of the central nervous system the toxin is altered by some unknown irreversible process to form a new toxic substance which circulates in the blood and which is not neutralized or affected by tetanus antitoxin, and which in sufficiently large quantities produces death. It follows then that once a lethal amount of toxin has been fixed by the central nervous system, no amount of tetanus antitoxin or other therapy will alter or affect the fatal termination of the disease. Since it is impossible to tell in any given case when a fatal amount of antitoxin-resistant toxin has been produced, this knowledge should in no way influence the treatment of any patient. It must be remembered that very severe symptoms and convulsions may result from less than a fatal amount of toxin.

If one accepts this concept of a constant progressive building up toward a fatal amount of antitoxin-resistant toxin, the importance of immediate administration of sufficient amounts of antitoxin by the most effective route becomes clearly apparent. At any hour or even minute the fatal amount of antitoxin-resistant toxin may be accumulated and the disease at that moment progresses from a curable to an incurable one.

For the initial treatment then in any case tentatively diagnosed as tetanus, the following steps are suggested in the order given. First, determine that the patient is not sensitive to the antiserum by a skin test. This can be read in fifteen minutes, during which time the antiserum can be prepared and the patient sedated if necessary. If the skin test is negative, 50,000 to 60,000 units of antiserum diluted in about 300 c.c. of normal saline solution should be started intravenously immediately and allowed to be absorbed in thirty to forty minutes. An equal amount may be administered intramuscularly at the same time. Should the skin test be positive, desensitization by the standard procedure must be carried out and usually two or more hours must elapse before intravenous administration of antiserum can be begun. This delay is regrettable, but unavoidable in the case of serum-sensitive patients.

Obviously no form of treatment is complete without a complete excision and eradication of the primary site of infection where the toxin is being constantly produced. This then should be the next step in the initial treatment. Under local or general anesthesia, as indicated, a complete and radical débridement or excision must be done whenever possible. In cases where multiple possible foci of infection exist or where no history of trauma is obtainable, the entire surface of the body should be inspected and all possible foci of infection excised or débrided. The further local treatment of these excised areas is probably adequately cared for by applications of some oxidizing substance such as hydrogen peroxide or zinc peroxide.

Firor[12] recommended an initial dose of 50,000 units of antiserum intravenously, 10,000 to 20,000 units locally around the site of infection prior to débridement, and 20,000 units intraspinally. He followed this by giving 5,000 units intramuscularly daily for eight to ten days.

At Charity Hospital during the last three or four years most of the tetanus patients have received 40,000 to 60,000 units of antiserum intravenously and an equal amount intramuscularly on admission. The subsequent administration has varied from repeating a similar dose on the second day with none thereafter to giving from 10,000 to 40,000 units daily for eight to ten days. It should be remembered that in cases where the foci of infection cannot be found or removed, much larger doses of the antitetanus serum should be continued daily for a longer period of time. Prior to about 1939, much smaller amounts of antiserum were customarily administered. Only 2 patients in the series received the antiserum intraspinally, both of whom lived, and only 11 patients received a local infiltration of antiserum around the wound site, of whom 4 died. Several authors have presented evidence that intraspinal administration of antiserum is not only without therapeutic value, but is probably harmful. Firor,[12] Jensen,[13] Caviness,[14] Weinstein and Beacham,[15] and Vener and Bower[6] have advocated the use of intraspinal administration and Aud,[16] Kirtley,[8] Dietrich,[9] and Boyce and McFetridge[1] have emphasized the high incidence of severe, and in many cases fatal, reactions, especially in children, following such intrathecal administration. It is rather difficult to conceive of the efficacy of intrathecal administration in view of the now quite conclusive evidence that tetanus toxin once fixed in the tissue of the central nervous system is no longer neutralized by antitoxin.

The importance of good nursing care of these patients cannot be overemphasized. The mortality of severe tetanus cases will not be appreciably decreased until each of these patients can be under constant supervision by a specially trained and experienced nurse.

In the further treatment of the patient there has been no routine procedure or set of orders. The following orders are presented as being fairly representative of the care that most of these patients received subsequent to the initial treatment:

1. Weigh patient
2. Chart intake and output
3. Keep gastric tube in stomach continuously
4. Provide special tube feedings every four hours
5. Give 3,000 to 4,000 c.c. of fluids daily
6. Turn patient frequently
7. Keep room darkened and quiet
8. Have only one attendant in the room at one time
9. Provide continuous nasal oxygen
10. Aspirate mouth and pharynx whenever necessary
11. Give ephedrine sulfate 1 per cent nose drops every three to four hours
12. Administer atropine, gr. $\frac{1}{150}$, every six to eight hours
13. Provide sulfadiazine, 1.0 Gm., every four hours
14. Give sodium bicarbonate, 2.0 Gm., every four hours
15. Use Trendelenburg position
16. Furnish avertin, 70 to 90 mg. per kilogram of body weight rectally every six hours for restlessness
17. Administer phenobarbital, gr. 2, every four hours as necessary to keep patient well sedated
18. Give sodium amytal, gr. 7½, intravenously as necessary for control of convulsions

The rationale of most of these orders is apparent. Atropine is given to aid in producing muscular relaxation as well as to decrease the respiratory tract secretions in a heavily sedated patient. The high calorie and high vitamin tube feedings are vitally important in the care of these patients. A patient who is having generalized muscular contractions is undoubtedly burning up a great deal of energy and must be provided with an intake of at least 3,000 to 4,000 calories daily. The administration of sulfonamides in therapeutic doses has become practically routine since 1941. This has been concomitant with a markedly decreased mortality rate. It is believed that the sulfonamides have an important role in lessening the mortality rate by helping to eliminate bronchopneumonia as a complicating cause of death. Of the 158 deaths in this series, 32 were recorded as being complicated by bronchopneumonia. It is highly significant that only 2 of these patients received any sulfonamide therapy and each of these 2 received the drug for less than thirty-six hours before death.

It is believed that the four important factors in decreasing the mortality rate are (1) early adequate amounts of antitoxin, (2) complete débridement of the focus of infection, (3) adequate sedation, and (4) sulfonamide therapy. As far as could be ascertained from studying the charts, nearly all these patients received fairly adequate sedation, attained principally by the use of avertin rectally and barbiturates orally, via stomach tube or parenterally. There was, however, considerable variation in the administration of the other three lifesaving measures. A detailed study of the effect of the administration or omission of these three measures on the mortality rate is presented.

Of the 158 deaths in the series, 59 occurred on or before the second day. As far as could be ascertained from these charts, the great majority of these deaths occurred within twenty-four hours of beginning of treatment plus the addition of a few others who died within thirty-six hours. It is believed that these patients may properly be considered as being moribund upon admission and that none of the known methods of treatment, however used, could have saved their lives. Not included in this group is 1 patient who died on the second day following an intravenous injection of curare. This leaves 158 minus 59, or 99 patients to whom the term of preventable or net mortality is applied in contradistinction to the previously mentioned moribund group.

An interesting observation concerning this moribund group is that only 63 per cent were Negroes, which is the same percentage as is the number of Negroes to the total group. This indicates that at least in this series, contrary to generally prevalent opinions, the Negroes did present themselves for treatment in approximately the same stages of the disease as did the white patients.

This leaves 293 patients who were not moribund upon admission, of whom 99 patients died, a net mortality of 34 per cent. Along with other therapeutic measures, 184 of these patients received débridement of the foci of infection, 65 of whom died, a mortality of 36 per cent. Seventy received sulfonamide therapy, of whom 22 died, a mortality of 31 per cent. One hundred twenty-nine patients received an initial dose of antiserum of 40,000 or more units intravenously and 20,000 or more units intramuscularly. Forty-two of these patients died, a mortality of 32 per cent. Of this latter number there were 46 patients who received a higher initial dose of antiserum amounting to 60,000 or more units intravenously and 40,000 or more units intramuscularly. Sixteen of these patients died, a mortality of 29 per cent. A still smaller number of 17 of these patients received 80,000 or more units intravenously, with 40,000 or more intramuscularly. Of this number 7 died, a mortality of 41 per cent.

There were only 39 patients in the series who received sulfonamides, débridement, and the minimal 80,000 or more units of antiserum on admission. Ten of these patients died, a mortality of 26 per cent. Of this latter group there were 20 patients who received the larger initial doses of antiserum, namely, 60,000 or more units intravenously and 40,000 or more units intramuscularly. Seven of these patients died, a mortality of 32 per cent. An even smaller number of this group of patients received an initial dose of 80,000 units or more intravenously and 40,000 or more units intramuscularly. Three of this number died, a mortality of 37 per cent.

These statistics, while not conclusive, indicate that there is a maximum effective initial dose of antiserum which is around 80,000 to 100,000 units, at least one-half of which should be given intravenously. Larger initial doses do not appear to have any appreciable effect on lowering the mortality rate, in fact, are associated with an increased mortality rate.

During the years of 1935 to 1940 there was a constant trend toward increasing the initial dosages of antitetanus serum in the treatment of tetanus. The patients in the series treated by Vener and Bower[8] and reported in 1941 received 200,000 units of antiserum locally, intramuscularly, intravenously, and intrathecally during the first twenty-four hours of treatment. The fact that they reported the lowest mortality of any large series cannot be overlooked. The excellent general care and nursing facilities accorded those patients, however, should be regarded as another factor in lowering the mortality rate. Caviness,[14] in 1943, recommended total doses as high as 300,000 to 550,000 units and many patients in this present series received as high total dosages. However, Firor[12] has shown quite conclusively that 50,000 units of the antitetanus serum intravenously will neutralize as much toxin as can be present in the blood stream. It would seem then that larger doses and subsequent intramuscular doses are indicated only as a safeguard against possible further production of toxin due to an incomplete or an impossible eradication of the foci of infection.

The total amounts of antiserum given are purposely omitted. Since the subsequent amounts given vary so widely according to the severity of the disease and the intervention of death in the fatal cases, it is believed that the initial doses are the only accurate criteria for a comparison of the therapeutic results.

Treatment of Nine Patients With Curare.—Some discussion has been carried on in recent years regarding the therapeutic value of curare in the treatment of tetanus. Under the supervision of Dr. John Adriani,[17] Director of the Department of Anesthesia at Charity Hospital, 9 patients in this series received intocostrin in an attempt to relax severe muscular spasms. The conclusions, in brief, were that curare has no effect on the course of the disease other than to relax muscle spasms temporarily, for one hour or less, and that use of the drug is accompanied by more hazards and toxic reactions than are justified in view of the temporary and purely symptomatic relief obtained.

Treatment of Five Patients With Penicillin.—Five of these patients received penicillin varying in total amounts from 250,000 to 600,000 units. Two of the patients died, a mortality of 40 per cent, both deaths occurring on the ninth hospital day. No beneficial effect of the drug on the course of the disease other than as a prophylaxis against secondary infection and pneumonia was observed in this small series of 5 cases.

SUMMARY

Three hundred fifty-two patients with tetanus were treated from Nov. 1, 1934, to Oct. 31, 1944.

The total mortality was 45 per cent. Excluding the deaths which occurred in the first thirty-six hours, the net mortality was 34 per cent.

The mortality has been substantially reduced in the last three years, during which time there has been a total mortality of 31 per cent, and a net mortality of 18 per cent.

Further reductions in the mortality rate may be obtained by adherence to the following reiterated factors of treatment:

1. Routine immunizations by tetanus toxoid
2. Routine administration of prophylactic antiserum (preferably 3000 or more units) in the treatment of all injuries to patients who have not previously received toxoid immunization
3. Education of lay people to report early and of doctors to recognize and institute immediate therapy for symptoms of early tetanus
4. Immediate intravenous administration of adequate amounts of antiserum
5. Complete excision or débridement of all possible foci of infection including umbilectomy in cases of tetanus neonatorum
6. Adequate sedation
7. Therapeutic doses of sulfonamides to prevent secondary infection and as prophylaxis against pneumonia
8. Daily administration of 40,000 to 50,000 units of antiserum intramuscularly to all patients in whom the original foci of infection cannot be found or completely removed
9. Good general nursing care
10. Adequate caloric and fluid intake by stomach tube feedings.

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GUNSHOT FRACTURES OF THE SHAFT OF THE HUMERUS

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DURING the North Burma campaign of 1943 and 1944, complete gunshot fractures of the shaft of the humerus comprised approximately 3 per cent of all battle casualties admitted to this general hospital. In comparison with gunshot fractures of other long bones, fractures of the humeral shaft were relatively simple to treat as far as the bone injury was concerned. The hanging plaster, introduced by Caldwell[1] in 1933 and widely used in civilian practice, proved to be ideal for the treatment of the majority of these fractures under conditions of warfare. Bone union in satisfactory position was obtained in almost every instance in which there was no significant bone loss. In spite of this, the high incidence of associated nerve injuries, the less frequent circulatory damage, and nonunion resulting from bone loss made this lesion an important military problem from the standpoint of rehabilitating the soldier and returning him to active duty. By expert treatment of these associated injuries, permanent disability may often be prevented.

ANALYSIS OF CASES

During the campaign, 100 patients with complete gunshot fractures of the humeral shaft were admitted to this hospital. Sixty of these were treated on the orthopedic section and were followed for a sufficiently long period to determine the final result of fracture treatment. These were consecutive cases and were unselected. Because of the exigency of the situation at various periods, the remaining forty patients were treated on the general surgical section. Since these were handled in a somewhat different manner, and because many were dispositioned to other hospitals before the final result was known, they are not included in this analysis.

Thirty-eight per cent of the fractures were in American and 62 per cent in Chinese soldiers. The "small arms" type of warfare in this campaign is shown by the missile causing the wounds: bullets in 62 per cent and shell fragments, usually mortar, in 38 per cent. The fractures were evenly distributed as to side and fairly evenly as to the segment of the shaft involved: 34 per cent in the proximal third, 19 per cent in the middle third, and 47 per cent in the distal third.

Ninety-three per cent of the wounds were débrided in forward hospitals, the average time of operation being 10.4 hours following injury. Débridement was performed within the first twelve hours in 69 per cent of all the injuries. Sulfanilamide was applied locally to every wound at the time of the primary operation, and oral sulfonamides were given to all patients before admission to this hospital.

In 80 per cent an abduction plaster spica was used for temporary immobilization during evacuation to this installation. Many of these were broken at the shoulder on arrival. In only 10 per cent was a hanging plaster used for immobilization during evacuation. The average interval before arrival at this hospital, after a litter haul of varying distances and an airplane ride of over 100 miles, was 3.5 days.

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Radial nerve paralysis was found in association with 33 per cent of the fractures, and median nerve paralysis in 7 per cent. There were no ulnar nerve injuries in this series. Evidence of circulatory insufficiency was present on admission in seven instances. In three the brachial artery had been injured. In the remaining four, constriction by an unpadded circular plaster, usually in the region of the elbow, was the cause of the ischemia. In no instance did gangrene result, but in two the ischemia was of sufficient duration to produce residual fibrosis and paralysis.

The hanging plaster was used for the definitive treatment of 80 per cent of the fractures. The remaining 20 per cent were treated by other methods (abduction plaster spica in 15 per cent, and skeletal traction with a Kirschner wire through the olecranon in 5 per cent) because of:

1. Bone loss of more than one inch (abduction spica)
2. Very high humeral fractures usually with associated injury to the shoulder joint (abduction spica)
3. Marked abduction of the proximal fragment (abduction spica)
4. Nonambulatory patients (skeletal traction)

The fractures were immobilized until there was x-ray evidence of bone union. This occurred on an average in 8.8 weeks. In four instances union occurred only after immobilization for 16 weeks.

Osteomyelitis occurred in five (8.3 per cent). In all cases it was localized and mild, and in no instance did it cause nonunion. Seven fractures had bone loss of more than one inch. The only five instances of nonunion (8.3 per cent) occurred in this group.

There were two deaths in this series. One was due to encephalitis of unknown etiology, and was unrelated to the fracture. At autopsy the fracture site was clean, and there was no evidence of embolism. Secondary hemorrhage three weeks after injury caused the second death.

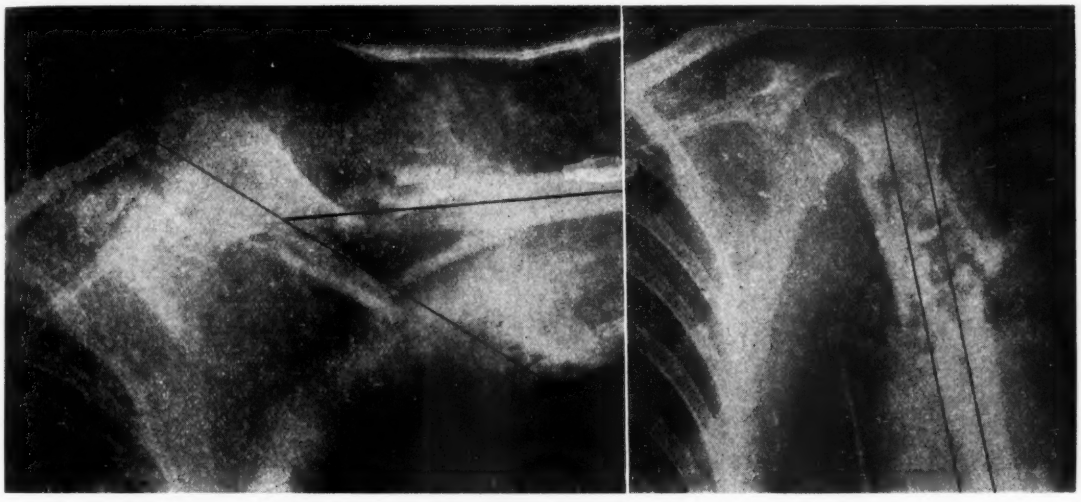
OBSERVATIONS

On the basis of our experience with the sixty gunshot fractures of the humeral shaft, certain opinions regarding their management have been formed.

Primary Treatment and Evacuation.—Although it is generally agreed that débridement should be performed as early as possible after injury, an interval of several days is not a contraindication to operation. Late débridement, from one to several days after injury, has appeared to be of value in preventing or limiting infection, and in shortening the period of wound healing. It seems probable that the establishing of free drainage and removal of unattached bone fragments, necrotic tissue, and foreign bodies, even in the presence of localized infection and inflammation, will aid in preventing the development of osteomyelitis. With the aid of sulfonamides, the danger of causing invasive infection by excising necrotic tissue in the presence of inflammation is minimal.

It is questionable whether the use of an abduction spica for evacuation of these fractures, as was done in 80 per cent of those admitted to this hospital, is justifiable. Many of the spicas had broken at the shoulder during transportation and, on arrival, these patients had considerable pain because of motion at the fracture site. Not infrequently wide abduction in a spica caused marked medial angulation. This could be corrected in almost every instance by merely dropping the arm to the side, as shown in Fig. 1.

Humeral fractures can be efficiently immobilized for evacuation in a hanging plaster. In patients who must travel in the recumbent position it is advisable to fix the plaster to the body with a few turns of the bandage. Advantages of the hanging plaster for evacuation are simplicity of application, saving of time, economy in the use of plaster, greater comfort for the patient, and, in most instances, better position of the fragments. The increased comfort of the patient when a shoulder spica was changed to a hanging plaster was impressive.



A. *B.*

Fig. 1.—Gunshot fracture of the proximal third of the humeral shaft. *A*, Position on admission in a 90 degree abduction spica; 37 degrees medial angulation. *B*, Perfect alignment obtained by merely dropping the arm to the side in a hanging cast. (Photograph by First Medical Museum and Arts Detachment.)

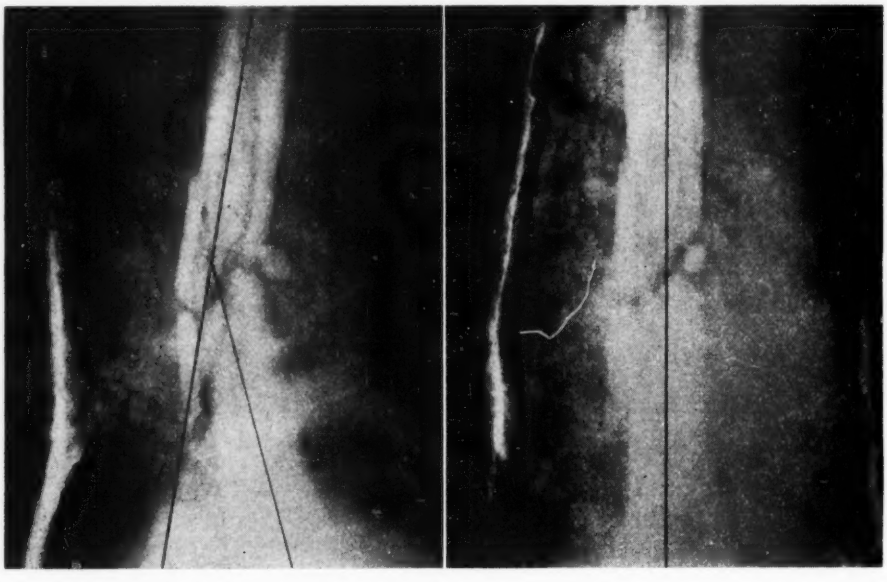
Treatment of the Fracture.—The characteristic displacements of the fragments found in simple fractures of the humerus were not often seen. External angulation in fractures of the proximal shaft was not common and abduction was rarely necessary to correct this deformity. Several fractures of the distal third were externally angulated. Pronation of the forearm as suggested by Böhler[2] was very helpful in correcting this (Fig. 2). In our experience it has not been necessary to keep patients treated with a hanging plaster in an erect or semi-reclining position during sleep for the first 10 to 14 days after injury, as recommended by Shands.[3] In Chinese patients, all of whom refused to sit up while sleeping, the efficiency of the hanging plaster in reducing the fracture and in maintaining good position of the fragments was not decreased. Whenever possible, the hanging plaster should be applied with the patient in a sitting position. If anesthesia is necessary, brachial plexus block is useful since the patient may remain upright.

The recent suggestion of Churchill[4] that the wounds of gunshot fractures be closed by delayed primary suture within ten days after injury has impressed us, for the early conversion of a compound fracture to a simple fracture is highly desirable. This procedure presupposes thorough débridement and a wound free from infection.

Nerve Injuries.—Because nerve injuries are responsible for most of the disability resulting from these injuries, their treatment is of greatest importance. It is now generally agreed that the highest incidence of nerve regeneration is obtained when repair is performed within the first few weeks after injury.

This necessitates close cooperation between the orthopedist and neurosurgeon. Whenever possible, nerves should be sutured at the time of delayed primary closure, if this is done.

Overzealous splinting of the wrist and fingers in extension for radial nerve injuries may result in fixation in this position. Splints for radial nerve palsy should be removable so that the wrist and fingers may be moved through their full range of motion several times daily. No splinting at all, with frequent active and passive joint motion, is better than continuous splinting. Suspension of the fingers and wrist by rubber bands to a "banjo" ring incorporated in the plaster is an excellent method of preventing deformities due to radial nerve injuries.



A. *B.*

Fig. 2.—Gunshot fracture of the distal third of the humerus. *A*, On admission with the forearm in supination; 25 degrees external angulation. *B*, Perfect alignment obtained by merely pronating the forearm. (Photograph by First Medical Museum and Arts Department.)

Circulatory Impairment.—The occurrence of circulatory embarrassment in seven instances emphasizes the importance of this complication. The commonest cause was constriction, usually at the elbow, by an unpadded circular plaster. Following splitting of the plaster, adequate circulation returned in all but one. In this instance there was residual partial paralysis of the hand due to the temporary ischemia. This complication can always be prevented by adequate padding and by frequent inspection for circulatory impairment by the personnel concerned in evacuation. At the first sign of ischemia the plaster must be split through its entire length and spread.

In three instances the brachial artery was injured. In two the artery had been ligated at the time of débridement and signs of ischemia were present on admission. Gangrene was feared in both, but, following elevation of the extremity and repeated blocking of the stellate ganglion with procaine, the ischemia disappeared and viable extremities resulted. In one there was moderate residual ischemic paralysis. In the remaining instance, arterial injury was not suspected until a secondary hemorrhage occurred on the tenth day after injury. This case emphasizes the fact that a palpable radial pulse does not exclude major arterial damage. Because the errors in management of this case are instructive, it is briefly described here.

CASE REPORT

A 27-year-old Chinese soldier was admitted in a plaster spica 3 days after a perforating bullet wound of the proximal humerus. He was in good general condition in spite of a hemoglobin of 8.0 Gm. The radial pulse seemed to be normal. Blood transfusions were given and a hanging plaster was applied. On the tenth day after the injury the wound bled an estimated 150 c.c. beneath the plaster. When the plaster was removed, no evidence of active bleeding was found, and the wound was not explored. After the wound remained dry for two days, the plaster was reapplied. Convalescence was then uneventful until the twenty-first day when, during the night, a sudden massive hemorrhage occurred from a divided brachial artery and death resulted before the plaster could be removed.

Secondary hemorrhage, no matter how minor, must always be considered an absolute indication for immediate exploration of the wound.

Osteomyelitis.—Osteomyelitis occurred in only five cases (8.3 per cent). In every instance it was well localized and of short duration, requiring only simple removal of sequestra to effect healing. It seems probable that early and adequate débridement was chiefly responsible for this. The routine use of local and oral sulfonamides may have been a factor. During the campaign penicillin was not available. Nonunion was not caused, nor was union significantly delayed, by osteomyelitis.

Nonunion.—In all fractures which resulted in nonunion, there was bone loss of more than one inch. In each instance a plaster spica was used for treatment, and efforts were made at the time of application to approximate the fragments. Two fractures, each with bone loss of two inches, united by the use of this method. In no instance in which there was a minor degree of bone loss did the traction of the hanging cast predispose to nonunion. It is entirely possible that fractures with bone loss of more than one inch might have bridged as well with a hanging plaster as with a spica. Fractures in which nonunion is inevitable should be recognized as early as possible in order that neighboring joints may be given physical therapy prior to the prolonged immobilization associated with bone grafting.

CONCLUSIONS

1. Our experience has shown that gunshot fractures of the humeral shaft can be adequately and comfortably evacuated in a hanging plaster to a fixed installation, and, in our opinion, this is the method of choice.

2. The hanging plaster is highly satisfactory for treatment of these fractures in most instances. Traction by the plaster does not predispose to nonunion in cases with minor degrees of bone loss.

3. Associated nerve injuries occur in a high percentage of these fractures, and are the most frequent cause of disability.

4. Ischemia due to constriction by circular plaster is a constant danger. It can be prevented by adequate padding and, if circulatory impairment is imminent, by splitting and spreading the plaster.

5. Secondary hemorrhage, no matter how minor, is an absolute indication for immediate exploration of the wound.

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RADIOGRAPHIC DIAGNOSIS OF HERNIA INTO THE LESSER PERITONEAL SAC THROUGH THE FORAMEN OF WINSLOW

Report of a Case

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(From the Hollenberg Clinic)

THE rare but interesting condition of hernia into the lesser peritoneal sac through the foramen of Winslow is reviewed at length by Ullman.* In his section under diagnosis he states that a preoperative diagnosis of hernia through the foramen of Winslow has never been made. My investigation of the literature confirms this statement. In his conclusion he asks, "Will a preoperative diagnosis of a hernia through the foramen of Winslow ever be made and will it be differentiated from other internal hernias?"

CASE REPORT

Mr. C., 76-year-old Scottish farmer, came to my office at 6 P.M., Dec. 13, 1933, complaining of acute intermittent epigastric pains.

Previous Personal History.—He always enjoyed good health until the year before admission, when he began to suffer recurrent attacks of diarrhea. His illness began at 10 A.M. the morning of Dec. 13, 1933. The pain in the epigastrium was, at first, mild and recurred at long intervals, but was becoming progressively more severe when he saw me at 6 P.M. No flatus or feces had been passed since the onset of the illness. The severe abdominal pains were localized to the upper part of the abdomen. There was also tenderness around the umblicus. During an acute attack of this colic he doubled up with pain, applying counterpressure to the abdomen with his hands. The attack lasted about two minutes and then gradually subsided. Examination of the abdomen revealed no bulging or any external hernia. The clinical picture was that of an acute obstruction. No peristaltic waves were visible during the attack of colic. Palpation over the epigastric region elicited excruciating pain. He had not vomited since the onset. There was marked epigastric resonance on deep percussion; there was also some fullness over the epigastrium. Being of the opinion that the obstruction was high up, I gave him barium and took the plate illustrated in Fig. 1. The lesser curvature presented two parallel crescentic outlines. It also showed the stomach hugging a rounded gas-filled mass. This led me to suspect that there was a hernia into the lesser peritoneal cavity, therefore the patient was sent to the Winnipeg General Hospital. There he was given four enemas with the hope of overcoming the obstruction, but with no avail. He was then given a barium enema. The result is shown in Fig. 2. The stomach still contained some barium; some of it had passed into the small bowel which appeared distended and pipelike, typical of a distal obstruction. The barium enema outlined the rectosigmoid descending and the left half of the transverse colon where the barium shadow abruptly ceased. The cecum ascending colon and right half of the transverse colon were not visible. There was a large, dark, rounded, gas-filled mass hugged by the stomach, and a gas distended bowel leaving this mass or being pinched off in the region of the foramen of Winslow could be seen. This confirmed the diagnosis of hernia of the cecum ascending and the right half of the transverse colon into the lesser peritoneal cavity through the foramen of Winslow. This diagnosis was recorded on the operative sheet before the abdomen was opened.

Operation.—Operation was performed at 10 P.M. that night. The abdomen was opened through a right upper rectus incision. On investigation, the stomach was found to be hugging a large tympanitic mass which was movable, tense, and balloonlike in consistency (Fig. 3). The gastrohepatic omentum could be moved over the mass freely. No cecum or ascending colon could be found. The tip of the appendix was located high up on the right side under the liver. The right half of the transverse colon was missing. Only the left half could be seen. When this was traced to the right it ended abruptly in strong adhesions to the gall

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*Ullman, Alfred: Surg., Gynec. & Obst. 38: 225, 1924.

Fig. 1.

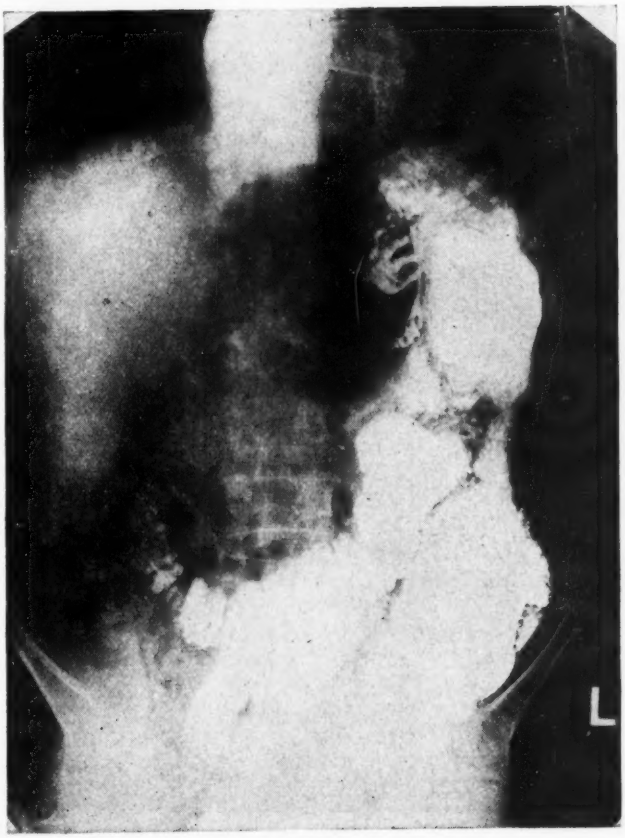

Fig. 2.

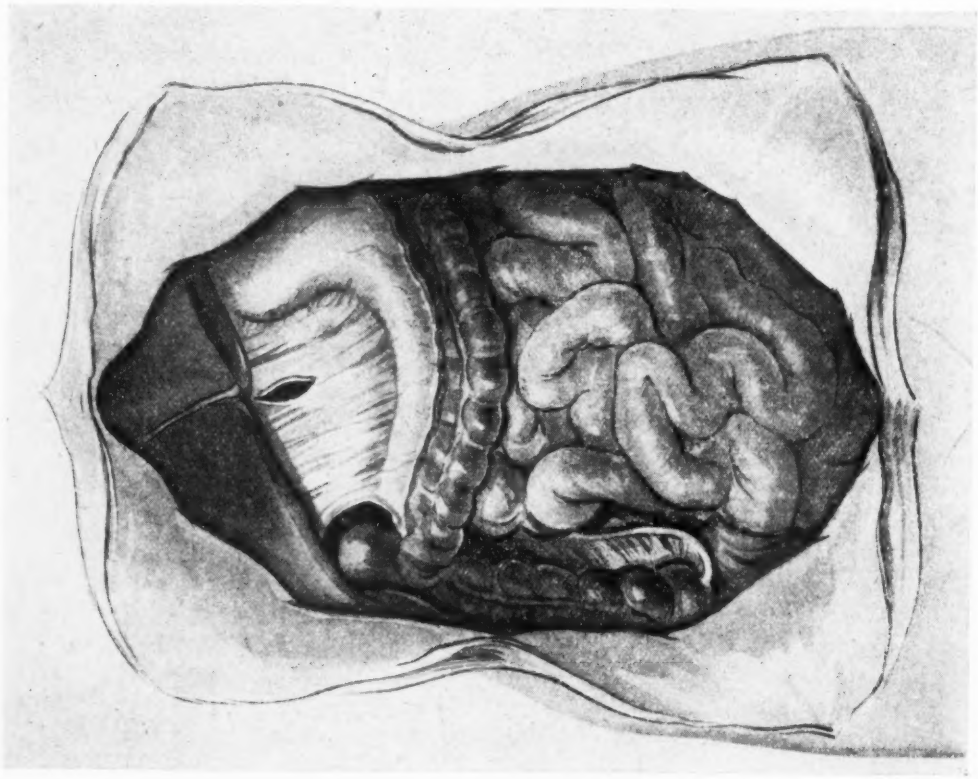

Fig. 4.

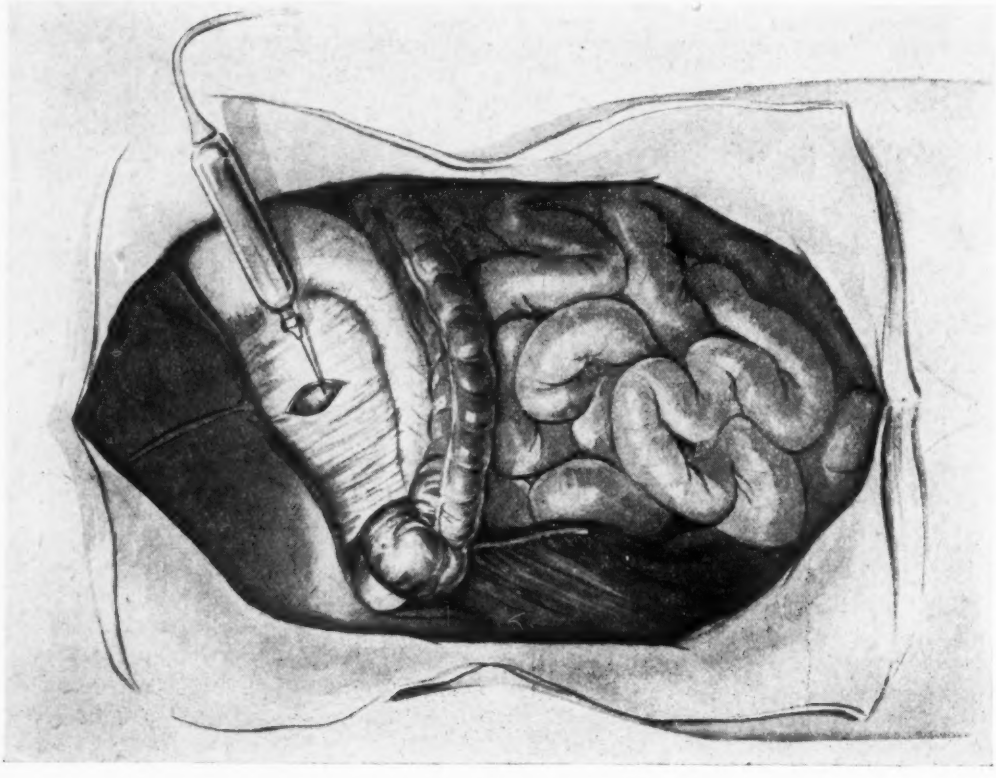

Fig. 3.

bladder and liver. This exploration confirmed the preoperative diagnosis. The left half of the transverse colon appeared to run straight into the liver. I attempted by traction on the colon and pressure over the lesser sac to deliver the incarcerated bowel without success. I then opened the lesser omentum and deflated the cecum by means of a needle attached to suction closing the hole in the cecum with a purse string and reinforcing it with Lembert sutures. The herniated bowel was then easily reduced through the foramen of Winslow (Fig. 4). The opening in the gastrohepatic omentum was closed. No attempt was made to close the foramen of Winslow. The cecum was tacked down in the right iliac fossa. The abdominal cavity was closed in the usual manner. The patient did well for two weeks. On December 27, his temperature became hectic, associated with chills. The pulse went up to 120 and respirations were 24. The left leg became swollen and painful. A diagnosis of thrombophlebitis of the iliac veins was made. His condition became progressively worse. There were no positive physical findings in the chest. He died December 29. The clinical diagnosis as to the cause of death was pulmonary embolism.

Fig. 5.

Autopsy Report.—Autopsy revealed a very thin man, 76 years of age whose left leg was very much larger than the right. It was not discolored. The pleural cavities showed no fluid. There were old adhesions over both lateral surfaces. The left lung weighed 525 Gm. There were apical scars. The lung was soft and crepitant throughout, showing quite marked emphysema. The main pulmonary artery contained fluid blood, but in three of the large branches there were firm masses of adherent thrombus. There were no infarcts in the lung. It showed only edema and congestion with some collapse in the posterior portion. The right

lung weighed 550 Gm. An apical scar and quite marked emphysema were present. There was a large adherent thrombus filling the upper portion of the branch of the pulmonary artery to the lower lobe. There was no infarct in the lower lobe, but there was a small one 1.5 cm. in diameter in the upper lobe just below the apex. The rest of the lung was congested, and there was some collapse of the posterior portion. There was no fluid in the abdominal cavity and the foramen of Winslow was large, admitting three fingers (Fig. 5). The right lobe of the liver was large and there was a transverse depression partially constricting off the lower half of the lobe. The gall bladder was covered with adhesions and was adherent to the transverse colon. The cecum was distended, unusually mobile, and was adherent to the omentum. The appendix was long, retrocecal, and tied down. The left iliac vein was filled with a firm adherent thrombus. The right vein was empty.

Summary of Autopsy.—Atheroma of aorta, pulmonary emphysema, pulmonary embolism, enlargement of the foramen of Winslow, and thrombosis of the left iliac vein. The thrombosis in the iliac vein was responsible for the embolus which lodged in the lung and caused death.

Probable Mechanism of Production.—The adhesions of the gall bladder and liver to the center of the transverse colon, and the fact that the right lobe of the liver was elongated a hand breadth below the costal margin, probably produced this rare hernia. The medial, spoon-shaped surface of the right elongated lobe of the liver directed the abdominal contents into the foramen of Winslow. The patient had recurrent attacks of diarrhea in the year before admission. These may be accounted for by the partial obstruction produced by the adhesions from the gall bladder across the middle of the transverse colon. The repeated attacks elongated the mesentery of the ascending colon until the right half of the transverse colon and ascending colon were forced into the lesser peritoneal sac and strangulated by the foramen of Winslow.

SUMMARY

The first case on record of hernia through the foramen of Winslow wherein a preoperative diagnosis was established from the radiologic investigation is presented.

EXTRADURAL ANESTHESIA

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CATHELIN, in 1901, was able to block the sacral and coccygeal nerves by injecting the anesthestic substance into the sacral canal, for peridural sacral anesthesia (caudal anesthesia) was limited to operations on the perineum. This led some surgeons to try the use of some different technical details in performing sacral anesthesia, so that they would be able to use it for operations in the abdomen. Larger doses as well as larger volumes of the anesthetic solution were employed, and the Trendelenburg position was tried in an effort to force the diffusion of the anesthetic solution to a higher level.

Extradural anesthesia, with the technique as used at present, was performed for the first time in 1920 by the Spanish surgeon, Pages, who also presented the first paper on this subject based upon forty-three observations with only two failures.[1] His researches were abandoned and forgotten after his death.

In 1931, at the Italian Congress of Surgery, Dogliotti described a "new method" of anesthesia to which he gave the name of "segmental peridural

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anesthesia,'' claiming for himself the priority of the discovery. In 1932, he presented his results to the American Congress of Surgery.[2]

Gutiérrez, in 1932,[3] called attention to the negative pressure of the extradural space (discovered in 1929 by Heldt and Maloney[4]) as a means of help in being certain of the exact location of the needle. Thus the ''sign of the droplet'' (also known as ''Gutiérrez's sign'') became known. Extradural anesthesia (the name given by Gutiérrez) has been also called ''anesthesia of Pages','' peridural anesthesia, segmentary anesthesia, epidural metameric anesthesia, or epidural anesthesia. Since my arrival in the United States in October, 1944, I have visited several hospitals, and I have not yet seen an operation performed with extradural anesthesia.

In 1938, when I spent some months working and studying surgery in Argentina, my attention was called to this method as used in the surgical service of Gutiérrez. I saw difficult operations performed with it, and during the postoperative course the patients felt none of the discomfort that results from anesthesia as it is usually given. I began a study of this new method of administering anesthesia and on my return to my own country put the knowledge to practical use in the hospital and with my private patients. An account of its effects in the first 100 cases was published in 1939 as a thesis, which I submitted as partial fulfillment of the requirements for promotion to the rank of Docente (Associate Professor) in the Medical School of the University of Brazil. Since then the number of cases involving the use of extradural anesthesia has grown steadily and I have observed its effects on more than 600 patients. I am a most enthusiastic believer in the value of this particular kind of anesthesia, because I am sure that it is one of the best available to the surgeon when he needs an anesthesia capable of long duration, but with as little toxic effect on the patient as possible. Before 1938, extradural anesthesia found little support among the surgeons of Rio de Janeiro. The few who had the occasion to try it considered it definitely inferior to the methods already in general use. I was happy to be able to call the attention of others to my excellent results and now many surgeons of Rio de Janeiro are relying on extradural anesthesia for serious operations.

I would like to express my views on extradural anesthesia and point out some of its results, based on my own case records.

I will not lose time with preliminary discussion of surgical anatomy or the physiology of the extradural (peridural) space, because such matters are well known. For reasons of clarity, however, I will divide my account into seven parts: Comparative study of extradural and intradural (spinal) anesthesia, technique, anesthetic solutions, locale (site) of puncture, evolution of the anesthesia, failures and accidents in its use.

COMPARATIVE STUDY OF EXTRADURAL AND INTRADURAL (SPINAL) ANESTHESIA

In intradural anesthesia, the anesthetic is injected into the subarachnoid space inside the duramater; in extradural, the solution is introduced into the extradural (peridural) space just outside the dura. The anesthetic injected into the extradural space does not come in contact with the cerebrospinal fluid, which obviates the danger both of toxic effect on the bulbar center and of resultant respiratory syncope. In extradural anesthesia the spinal ganglia, the sympathetic and spinal nerves, and the dura associated with the radicular nerves are anesthetized.

The changes of blood pressure after extradural anesthesia have not the same dangerous aspects that they have with intradural. Arterial hypotension, the dread of anyone giving intradural anesthesia, occurs much less often with the extradural method. In 600 cases, with blood pressure determinations made immediately before and after administration of the anesthetic and supportive drugs, at the beginning and end of the operation, and during twenty-four to forty-eight hours in the postoperative course, I made the following observations: In 66 per cent, the blood pressure rose; in 25 per cent, it fell; and in 9 per cent it showed no alteration. When the pressure falls, I use a supportive drug (ephedrine, 0.05 Gm.). This may also be used prophylactically.

The duration of the extradural anesthesia is longer, its range of action is wider, and accidents during and after its administration occur less frequently than in anesthesia of any other kind. Using an average of 45 c.c. of a 2 per cent solution of novocain, some patients have had anesthesia lasting more than four hours, but the average duration ranges from two to three hours. After this period of surgical anesthesia there is relatively long absence of sensitivity, very profitable for the patient because he does not feel pain in the postoperative period. Another great advantage of extradural anesthesia is the rarity of post-anesthetic reactions, such as headache, vomiting, and urinary retention. Late postoperative reactions, even after prolonged anesthesia, are likewise reduced. All these complications are quite usual after intradural anesthesia.

A modern and efficient surgeon must know every type of anesthesia, practically as well as theoretically, because each has its peculiar indications. A thorough examination of the patient and, based on that, the selection of the kind of anesthesia best for the particular case, should eliminate failure and accident on the part of the surgeon and anesthetist. Accidents during or after extradural anesthesia depend somewhat on the skill of the anesthetist. It need hardly be remarked that in nervous and anxious patients, in those in a malnourished, cachectic, or septic state, and in those with spinal deformities or ossifications of the interspinal ligaments or ligamenta flava, this method is not effective.

TECHNIQUE

The extradural space can be reached by the use of either of two techniques, the indirect or direct punctures. In the first, the needle is advanced until it perforates the duramater, which is indicated by the reflux of the cerebrospinal fluid. The needle is then withdrawn slowly until there is no reflux of the liquor and the point rests in the extradural space, where the anesthetic is injected. By the direct process, the anesthetist tries to reach the extradural space without perforating the duramater. Each time that he fails to do so, and perforates the dura, he withdraws the needle and makes another puncture in another interspinal space. This direct process is more correct and less dangerous, because with it there is no possibility that the anesthetic might penetrate the subarachnoid space through the opening in the dura.

The puncture is made with a special needle (the best is Gutiérrez's needle) in the area between the seventh cervical and fifth lumbar vertebrae, as required, with the patient in a sitting position. There are several signs to indicate that the needle is in the extradural space, but the best is the "sign of Gutiérrez" or the "sign of the droplet," which is based on negative pressure in the extradural space. After introducing the needle, the anesthetist progresses slowly, watching for the "sign of Gutiérrez." After passing through the superficial planes, with

the special needle, the anesthetist withdraws the stylet from the needle and fills the latter with the anesthetic solution in such a way that he leaves a drop of the solution hanging from the outer receptacle of the needle. He then advances the needle, pushing as slowly as possible, and when the epidural space is reached, the drop is aspirated inward. In some cases, there is only an oscillation of the drop, but usually a perfect suction is observed. With this sign, and the assurance that the point is in the extradural space, 10 c.c. of air are injected to separate the dura from the end of the needle. The anesthetist then injects 5 c.c. of anesthetic solution and waits five to ten minutes. The latter precaution is essential to ascertain whether the dura has been punctured. If it has, the specified amount of anesthetic is sufficient to produce intradural (spinal) anesthesia. If, after this time, no signs of anesthesia are noted, the rest of the solution is injected. The amount of the anesthetic solution depends upon its concentration and the duration of the operation.

Aside from the sign of Gutiérrez, the anesthetist may be guided by the sensation of having penetrated the ligamentum flavum and by the lack of resistance to the injection of the solution when the needle is in the extradural space. The distance between the skin and the extradural space varied from 3 to 7 cm. The sign of Gutiérrez was positive in 89 per cent of my observations. When the sitting position cannot be maintained, the patient may remain in the lateral position. However, in the latter position the technique is more difficult.

ANESTHETIC SOLUTIONS

The drugs used for extradural anesthesia are many. The concentration of the solution should be the same as a 2 per cent solution of novocaine. I have experimented with several solutions, including 1 per cent and 2 per cent novocaine (procaine), a 2 per cent solution of scurocaina, a 2 per cent solution of pantocain (pontocaine), a 0.1 per cent solution of percaine (nupercaine), and "Gutiérrez' mixture." Of the various solutions used for extradural anesthesia, Gutiérrez' mixture (0.5 neotutocaina [pontocaine] and 0.50 novocaine [procaine]) has proved the most satisfactory. A very important matter, if the anesthetist is to avoid failure, is the sterilization of the anesthetic mixture. Novocaine changes easily under heat, and the sterilization is delicate. Gutiérrez' mixture must be sterilized by tindalization (fractional method) for one-half hour, four to five times, in a maximum temperature of 70° C.

The anesthetic solution is dissolved in 50 c.c. of physiologic saline solution, and ten drops of adrenalin added. The adrenalin produces vasoconstriction in the extradural venous plexus, delaying the absorption time of the anesthetic and prolonging its duration. Immediately after the anesthesia is produced, 3 c.c. of caffeine are injected subcutaneously to prevent vomiting and nausea from the novocaine intoxication. An interval of twenty minutes is desirable between injection of the anesthetic agent and beginning the operation.

In my series, the amount of solution ranged from 20 c.c. for appendectomy in a child to 46 c.c. for a gastrectomy which lasted two hours and forty-five minutes.

POINTS OF PUNCTURE

For operations in the neck and thorax, the puncture is made between the seventh cervical and first thoracic vertebrae. A puncture between the eleventh thoracic and third lumbar vertebrae produces anesthesia for operations in the genitourinary field and on the sympathetic nerves and digestive tract. To anes-

thetize the perineum and lower extremities, the puncture is made between the third and fifth lumbar vertebrae. In such cases the patient is left sitting for fifteen minutes after the injection.

DEVELOPMENT AND DURATION OF THE ANESTHESIA

The effect of extradural anesthesia is not as immediate as that of intradural. Ten minutes after injection there is a large zone of anesthesia which continues to enlarge for about twenty minutes, and sometimes extends from the chin to the feet. The diffusion of the anesthesia is variable, but generally it depends upon the concentration of the solution, the amount injected, and the point of puncture. Likewise, its duration depends upon the strength of the anesthetic, the substance used, and the dose injected. The most successful results, both of development and duration, in my experience, were obtained with Gutiérrez' mixture. With an average dose of 40 c.c. of anesthetic solution, I have had anesthesia for two and one-half hours or more.

As stated earlier, extradural anesthesia is indicated for operations in the neck, thorax, abdomen, and extremities, and causes few postoperative complications, but it is contraindicated in nervous or anxious patients, in those in a malnourished, cachectic, or septic state, and in those with spinal deformities or ossification of the interspinal ligaments or ligamenta flava.

For some time I have been using, with very good results, a sedative medication just after finishing the anesthetic injection.

My experience with the effects of extradural anesthesia on children is limited, since I have had only two cases. I have, however, records of its successful use on fifteen patients older than 70 years, and on one 85 years of age.

FAILURES AND ACCIDENTS

Failures and accidents with extradural anesthesia are due largely to inexperience and improper techique. The skill of the anesthetist is a determining factor as is occasionally, also, the quality and amount of the anesthetic solution. Syncope may occur during induction, although no such reaction occurred in my series. At most, there was some nausea in a few patients. Owing to insufficient concentration of the anesthetic in a few cases at the beginning of the series, supplementary general anesthesia was required, and in a few others, because of the patient's extreme nervousness, a different form of anesthesia had to be resorted to. There was only one complete failure of anesthesia.

CONCLUSIONS

1. Extradural anesthesia is one of the most satisfactory, because it meets many of the requirements of a good surgical anesthesia with a minimum of dangerous effects to the patient.

2. An important recommendation for extradural anesthesia is the rarity of postanesthetic reaction.

3. Late postoperative reactions, even after prolonged anesthesia, are likewise reduced with this method.

4. The "sign of Gutiérrez" or "sign of the droplet" is the best indication that the needle is in the extradural space.

5. Injection of the anesthetic solution must be made as slowly as possible, and always in fractionated amounts.

6. Twenty minutes must elapse after injection before the surgeon may begin the operation.

7. Accidents during or after anesthesia depend somewhat upon the skill of the anesthetist.

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SEMIELASTIC COTTON GAUZE BANDAGE FABRIC

CHARLES F. GOLDTHWAIT,* AND JAMES H. KETTERING,* NEW ORLEANS, LA., AND COMMANDER MOORE MOORE, JR.,† MEDICAL CORPS, U. S. N. R.

THERE has been a long-felt need for a surgical gauze with a moderate amount of elasticity so that it will stretch or "give" while being applied and so exert moderate pressure. A gauze has recently been developed which has these plus other unusual and useful properties. It has stretchability and elasticity which make it somewhat self-fitting and self-tightening; the elasticity, together with a nonslip surface texture, causes a bandage made from it to cling rather than to tend to loosen while in place. The new fabric is all cotton, is in effect a preshrunk gauze, and has no similarity to conventional elastic bandages.

Bandages made from the new gauze have been submitted to several hospitals for clinical tests, in some instances by request. Reports have been very favorable, but the number of requests for additional quantities constitutes the best evidence that this bandage meets a real need. It has been on trial for over one year to the extent of about 9,000 unit strips, more than half of which have been used at the U. S. Naval Hospital in New Orleans where this bandage has supplanted all others for securing dressings about freshly operated joints.

This new type of bandage has properties of general interest, as well as specific advantages, which have recommended it for continued use at the Naval Hospital, especially in orthopedic surgery, and for various other purposes. First, it meets the general requirement that it can be sterilized by any of the standard recognized techniques without its special characteristics being affected. Next, its light weight is also of general interest because a bandage of given bulk in terms of thickness may weigh one-fourth less than one of ordinary gauze of 44 by 36 thread count.

The superior ability of this bandage to conform to the contours of the body and to remain in place makes it ideal for securing splints of all types.

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†U. S. Naval Hospital, New Orleans, La. The opinions or assertions contained in this article are the private ones of the writer and are not to be construed as official or reflecting the views of the Navy Department or the Naval Service at large.

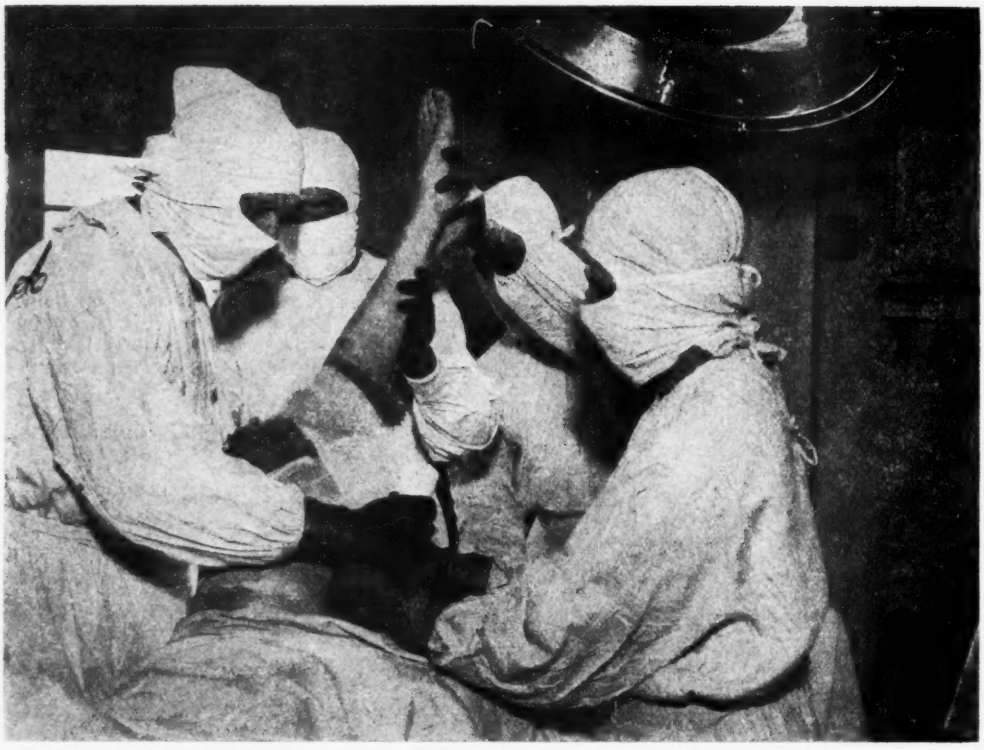

Fig. 1.—Application demonstrating special properties of semielastic gauze.

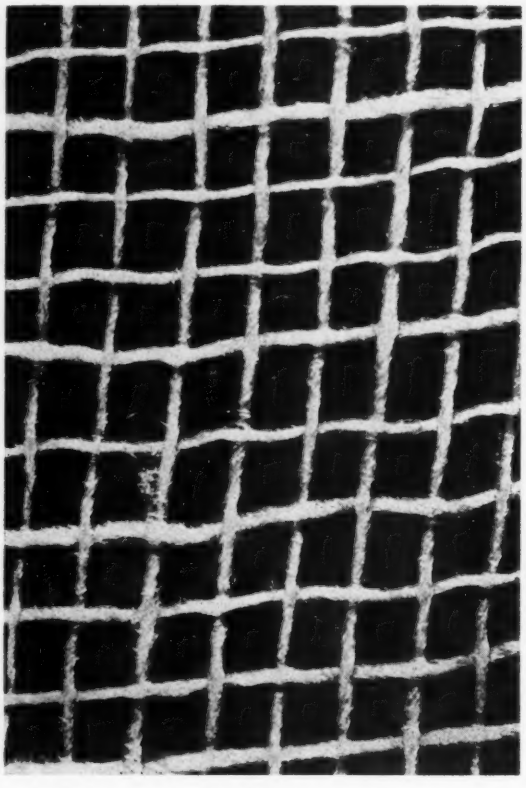

Fig. 2.

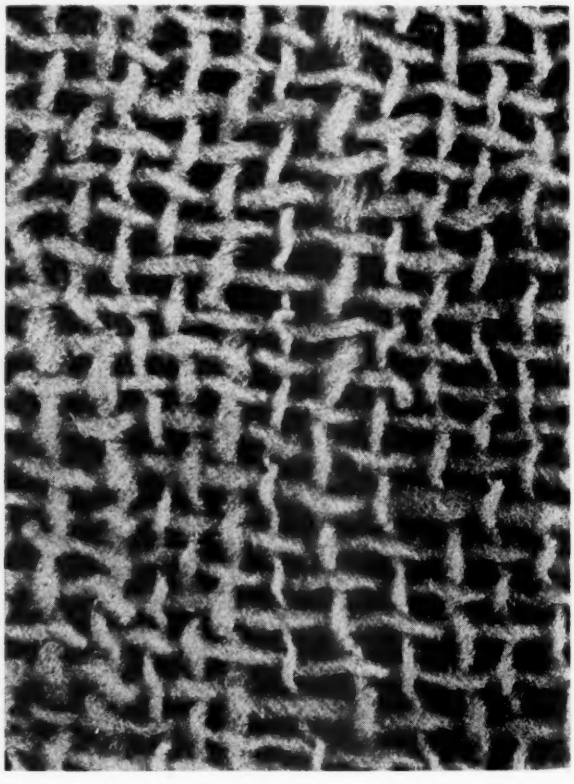

Fig. 3.

Figs. 2 and 3.—Ordinary gauze and semielastic gauze made from it. Views enlarged to the same scale.

The bandage can be used in conjunction with plaster without fear of "drawing" in any plane. In fact, its preshrunk character allows use either over or under plaster without danger of necrosis from cutting into the skin after drawing. It has proved very satisfactory for binding plaster splints and does so without adding appreciable weight to them. The bandage is unusually suitable for pressure dressings including use for burns, varicosities, and skin grafts. It is particularly good about joints where it is most desirable that the bandage should not slip. It should be emphasized that a steady even pressure can be applied easily and maintained for a prolonged time with this bandage. This is true whether there has been moderate swelling or whether swelling has subsided subsequent to bandaging. Because of its elastic properties and clinging power, the bandage tends to remain in situ, which is imperative about joints when the patient is ambulant or is permitted or encouraged to use the joint.

The only defect of any consequence noted in over one year's trial has been a mild tendency to gather on the short side of a joint when used in applying a spica type bandage. However, this is no more so than in conventional types and any mild tendency to chafe can be readily controlled by placing a pad in the axilla or groin. With these bandages, there has been noted also a tendency to gather about the torso or chest, but this can be largely controlled by the use of transverse adhesive strips at the time of application.

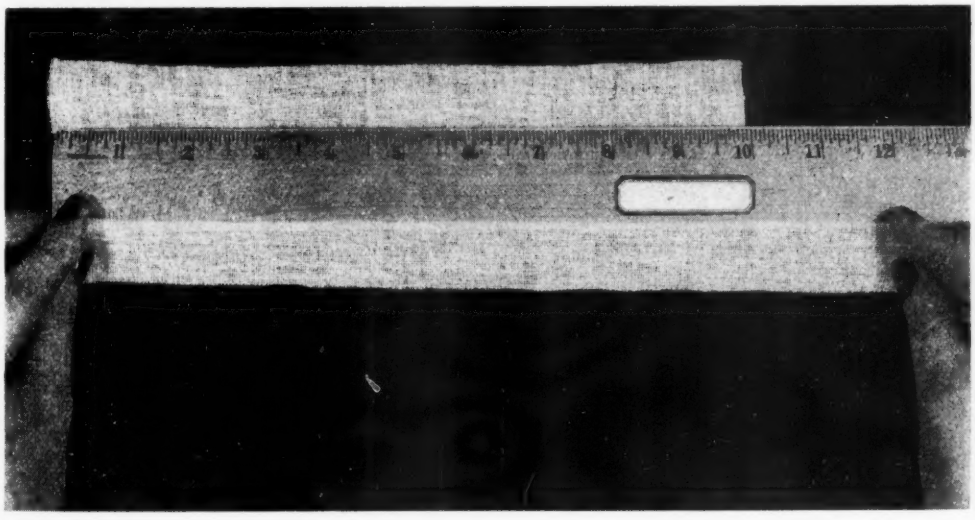

Fig. 4.—Semielastic gauze easily stretches 20 per cent.

This bandage fabric is an outgrowth of the well-known fact that immersion in concentrated sodium hydroxide solution causes cotton to shrink, primarily because it swells the fibers. The tendency of the swelling fibers to expand within the spiral structure of the yarn in the cloth causes the yarn to shorten which, in turn, causes the cloth to shrink. If the material is of suitable open weave like the lighter weights of surgical gauze, pronounced crimps and kinks are also formed which increase the shrinkage but make the goods easily stretchable and somewhat elastic. The complete effect is shown quite clearly in Figs. 2 and 3, which represent the original and treated gauze on the same scale. The shrinking, or mercerizing treatment, as it may be called, causes the yarn to expand in diameter and to become loose in construction, to shrink until the interspaces in the cloth are much smaller, and to form many crimps and kinks, some of which do not show because they are standing vertically to the plane of the fabric.

The ultimate result is that the cloth stretches easily under low tension, due to straightening of the yarns and tightening of their structure, but returns elastically, when released, to approximately its dimensions in the shrunk form. Most of the gauze prepared in this manner, if stretched 20 per cent, for example, from 10 inches to 12 (Fig. 4), has an average recovery of 15 per cent, to a new length of 10.5 inches. The elastic properties represented by these figures have proved very satisfactory for all around use. The surface irregularities, which are due to crimping and kinking, intermesh as each layer of a bandage or dressing is added, so that no layer can slip upon the others. Thus the bandage is interlocked, does not come apart readily, and also tends to stay in position.

The fabric as used in most of the clinical trials has been somewhat heavier than standard bandage material, but fewer layers are used. The weight can be modified by suitable selection of the initial gauze. Up to the present time the bandages have been made only on a rather large laboratory scale, but their special properties and general utility suggests that they be made available commercially.

COD-LIVER OIL OINTMENT IN SURGERY

Topical Application

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COD-LIVER oil ointment has been used in this country for more than a decade and over a much longer period in Europe. Many who have tried it have continued its use. The purpose of this paper is to present the results obtained from the extensive use of cod-liver oil ointment in a large series of cases consisting of a variety of lesions.

REVIEW OF LITERATURE

In 1934, Löhr[17] published his original paper presenting a series of cases, consisting of burns and a wide variety of clean and infected wounds treated with cod-liver oil applied topically. In the original and subsequent reports,[18-20] Löhr was enthusiastic and convinced of its superiority in the treatment of many surgical lesions. He was soon aware of the rapid liquefaction of nonviable tissue, its stimulating effect upon healing, and the minimal pliable scarring following its use. Löhr found cod-liver oil to be bacteriostatic and bacteriocidal.

Steel[30] found the cod-liver oil treatment to be very satisfactory for burns. He was particularly impressed by the reduction of pain. In 1936, Davson[6] presented experimental evidence to the effect that cod-liver oil definitely stimulates the healing process. He found an increased infiltration of the surrounding tissues with scavanger and repair cells.

In 1931, Thompson and Sheard[31] reported irradiated oil to have a decided bacteriostatic action. Puestow, Poncher, and Hammatt[27] treated experimental burns with tannic acid and cod-liver oil ointment. They found the healing time

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to be 25 per cent less when the latter was used. Kozanjian,[14] Tumanskiy and Yatsevich,[32] Pyke,[28] Daver,[5] Mutschler,[24] Salzmann and Goldstein,[29] Iost and Kochergin,[12] Lauber,[15] Proto,[26] Hayashi,[10] Lichtenstein,[16] Abramowitz,[1] Aldrich,[2] Heald,[11] Brandaleon,[3] Clayton,[4] MacCollum,[22] and many others recommended the cod-liver oil ointment treatment for a wide variety of surgical lesions. Their observations correspond to those previously cited.

In 1941, Hardin[7] published an excellent paper, giving a comprehensive review of the literature and presenting 346 additional cases in which the lesions were treated with the vitamin ointment. He reported the first cure of "Meleney ulcer" by the topical application of the ointment. Hardin recommended it as a "superlative method" of local treatment for burns and clean and infected wounds.

ACTIVE AGENTS AND EFFECTS PRODUCED

There is not complete agreement as to the active agents in cod-liver oil ointment or exactly what reaction takes place when it is applied topically to wounds. Lauber[15] and Lundh[21] concluded that vitamins A and D are absorbed and act systemically. Drigalski[33] and others thought vitamin A to be the active agent. Many are convinced that both vitamins A and D are essential and in order to produce the most satisfactory effect, they must be in the same proportion as that found in crude cod-liver oil. A few investigators believe the beneficial effect is due to the fatty acid content of the ointment. There is evidence that unsaturated fatty acids are related to the health and healing power of the skin. It can be stated with certainty that the same beneficial effect is not produced when the vitamins are given orally. There is probably an increased local vitamin requirement which is met by the topical application of the ointment.

Despite the disagreement concerning the active agents and the effects thereby produced, we know that cod-liver oil ointment is sterile, bacteriostatic, mildly bacteriocidal, and nonirritating; that it decreases pain, aids in liquefaction of dead tissue, and forms a thin pliable protective membrane on wound surfaces to which it is applied. Lauber[15] presented experimental proof that a stimulating effect is produced upon epithelial and connective tissue cells. Clayton[4] stated that cod-liver oil ointment produces an elastic membrane by interfascial precipitation of colloids from their aqueous state. It does not precipitate or "fix" proteins and therefore does not damage the remaining viable epithelial cells. This form of treatment decreases the necessity for skin grafting but when necessary it can be accomplished early because the ointment produces a clean granulating surface.

TABLE I. CLASSIFICATION OF 713 LESIONS TREATED WITH COD-LIVER OIL OINTMENT

WOUND		NO.
Burns		171
Major, treated with C.-L.O. ointment as the only local treatment	26	
Major, treated with C.-L.O. ointment secondarily	20	
Minor	125	
Denudation and avulsion type		307
Cutaneous and deeper structure defects following surgical procedures		101
Osteomyelitis		9
Acute	4	
Chronic	5	
Sloughing and draining abdominal		19
Chronic ulcerations (5 tuberculous)		24
Miscellaneous clean and infected		82

CLINICAL APPLICATION

In this series, 171 burns were treated by the topical use of cod-liver oil ointment, crude cod-liver oil, Gadoment,* and Vitamin A and D ointment.† Forty-six cases were of major severity. Twenty-six burns were treated with cod-liver oil ointment topically, as the initial and only form of local therapy. Twenty were treated with the ointment secondarily and after other forms of local therapy had proved unsatisfactory. Eschar-producing agents had been used as the initial form of local therapy in the majority of the twenty cases.

In many of the cases it was necessary to administer plasma and other familiar systemic measures before any consideration was given to local burn therapy. Treatment of the burned lesion itself was always considered of secondary importance and was carried out only after instituting treatment for shock or for its prevention.

Early in the series the more severe burns were thoroughly debrided under anesthesia but the majority were less thoroughly debrided following the administration of analgesics. After aseptic débridement and thorough flushing of the burned surfaces with warm saline solution, they were covered with cod-liver oil ointment-impregnated fine-mesh gauze. Firm or pressure dressings were then superimposed. Extremities readily lend themselves to pressure dressings, while firm dressings are as much as can be accomplished when other portions of the body are involved. Mechanic's waste proved a satisfactory form of pressure dressing. It is cheap and easy to sterilize. Infrequent change of dressing is recommended, as redressing often admits secondary infection. It is usually not necessary to change the original dressing in less than seven days and it may well be left untouched for much longer. Properly managed burns do not often become infected.

The twenty-six major burns receiving cod-liver oil ointment as the only form of local therapy responded favorably to the treatment as outlined. Many seemingly important observations were made on the patients during the course of treatment. They required considerably less sedation than patients treated with eschar-producing agents and noticeably less than those treated with petrolatum gauze. Infection was almost nil in those treated with cod-liver oil ointment and petrolatum gauze, while it was present to some degree in practically every case in which eschar-forming agents had been used. The healing time was ten days longer in the group in which tannic acid was used and four and one-half days longer in the one in which petrolatum was used. Scarring was considerably less and there were no contractures on the patients treated with the vitamin ointment. Severe scarring and frequent contractures were present in the group treated with eschar-forming agents. The explanation is that the eschar-producing agents destroy remaining islets of viable epithelial cells, which would act as regeneration centers, while cod-liver oil ointment produces no further injury to those cells.

In all fairness it should be added that it is very difficult to evaluate impartially all factors encountered in treating clinical burns. In many cases it is impossible to distinguish between second and third degree burns when first seen.

There was no greater need for plasma than in the control groups of twenty similar cases which the patients were treated with petrolatum gauze and twenty treated with tannic acid and other eschar-producing agents. Despite the claims

*Produced by the E. L. Patch Company, Boston, Mass.
†Produced by White Laboratories, Inc., Newark, N. J.

of those who favor eschar-producing agents, the loss of fluid from the burned surface is slight and of little importance as compared with the loss of fluids into the local and the uninvolved tissues. That has been substantiated by several previous writers.[8, 9, 13, 23, 25] The diffusion of plasma into the tissues is the important factor leading to hemoconcentration and shock. At least theoretically, the ointment combined with pressure dressings would decrease the diffusion of plasma, thus reversing claims made by tannic acid advocates.

TABLE II. ANALYTIC COMPARISON OF TANNIC ACID AND COD-LIVER OIL OINTMENT IN THE LOCAL TREATMENT OF BURNS

COMPARATIVE POINTS	TANNIC ACID	COD-LIVER OIL OINTMENT
Control of pain	Questionable value	Definite value
Range of use	Limited	Applicable to entire body
Protective covering	Rigid eschar	Pliable membrane
Infection	Frequent	Infrequent
Scarring	Moderate to severe	Minimal to moderate
Contractures	Frequent	Absent
Dressings	Not necessary	Infrequent changes
Skin grafting (3rd degree burns)	Frequent	Infrequent
Fixation of tissue	Deep fixation with destruction of viable cells	None
Healing time	Prolonged	Shortened
Pressure dressings	Not applicable	Very applicable
Primary shock	No effect	No effect
Secondary shock	No effect	Of apparent preventative value
Untoward reactions	Not uncommon and may be very serious (liver necrosis)	None

Three hundred seven denudations and avulsion type wounds were treated by the topical application of cod-liver oil ointment or packing with ointment-impregnated gauze. Many of the wounds were rather severe, involving large areas of skin and subcutaneous tissues, and a number presented open muscle wounds. Due to loss of tissues it was impossible to close some of the wounds while others were grossly contaminated, contraindicating primary closure. The wounds were thoroughly debrided, flushed with a large quantity of warm saline solution, and cod-liver oil ointment was used as the only form of local therapy. Otherwise the patients were treated in the usual manner. The dressings were changed infrequently. Wound infections were uncommon and no severe infections were seen. The defects filled with healthy granulation tissue in a surprisingly short time. Healing time was five to seventy-five days, with an average of seventeen days for the group. Scarring was minimal to moderate. This form of treatment proved entirely satisfactory.

One hundred one cases with cutaneous and deeper structure defects, following surgical procedures, were treated in a similar manner. The wounds ranged from small cutaneous defects resulting from removal of moles to large deeper structure defects resulting from excision of pilonidal cysts and amputation of breasts and extremities which for various reasons could not be closed at the time of operation. The wounds were treated with the ointment gauze as the only form of local therapy. Infection did not develop in the clean wounds and quickly subsided in the infected ones. All wounds healed satisfactorily without skin grafting. Again, the surprising feature was the rapidity with which the defects filled with granulation tissue thus facilitating more rapid epithelization.

The cod-liver oil ointment treatment was used in nine cases of osteomyelitis. As recommended by Löhr,[20] the involved areas were widely incised, the pus evacuated, sequestra removed, wounds loosely packed with cod-liver oil oint-

ment gauze, large absorbent dressings applied, and the extremity immobilized in a plaster cast. No windows were cut in the casts. The casts were not removed or reapplied for approximately three weeks and in some instances the original casts remained undisturbed much longer. The treatment was satisfactory in this small series of four acute and five chronic cases of osteomyelitis of extremities.

Nineteen sloughing and infected abdominal wounds were treated by packing or injecting with cod-liver oil ointment. Three of the patients presented the typical sloughing and the undermining postoperative abdominal wounds, following appendectomy for perforated gangrenous appendicitis. Many forms of local treatment had been applied in each instance before resorting to cod-liver oil ointment. The wounds were either filled with the ointment or packed with impregnated gauze. In each instance healthy granulation appeared in the bottom of the wounds in five to seven days. All healed in four to nine weeks. Several other chronically infected abdominal wounds were treated in a similar manner with good results.

The vitamin A and D ointment treatment was used in a large variety of miscellaneous surgical lesions, such as: tuberculous ulcerations and sinuses, trophic, varicose, and other chronic ulcerations, large abscess and carbuncle cavities, frostbites, x-ray burns, donor and recipient skin grafting sites. It proved of particular value when applied to donor and recipient skin-grafting sites. The ointment gauze dressing can be changed easily without disturbing the delicate graft. Let it again be emphasized that dressings should be changed infrequently.

SUMMARY AND CONCLUSIONS

A series of seven hundred thirteen cases is presented. One hundred seventy-one were burns, with the remainder representing a wide variety of surgical conditions. Twenty-six burns of major severity were treated with cod-liver oil ointment as the only form of local therapy. The results were considered superior to results in similar groups in which treatment was with eschar-producing agents and petrolatum.

Cod-liver oil ointment is of real value in the treatment of burns and many other surgical lesions. This method of treatment has all the advantages of tannic acid in burn therapy but eliminates many of the disadvantages. It is nontoxic, nonirritating, and applicable to all portions of the body. The ointment is bacteriostatic, bacteriocidal, speeds up liquefaction of devitalized tissue, and stimulates epithelial and connective tissue growth. Minimal pliable scars result. The preparation is sterile, easy to prepare in the crude form, not expensive, and may be obtained on the market as a refined odorless product. No additional equipment is needed and no skill is required in order to apply this form of treatment.

Cod-liver oil ointment is presented as a simple and effective local dressing with a wide range of use.

Space does not permit detailed case reports which appeared in the original thesis.

The author is deeply indebted to Dr. W. A. Coventry, Dr. T. O. Young, and Dr. E. L. Tuohy for their help in the preparation of this paper.

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CARE OF THE INJURED EXTERNAL EAR

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INJURIES to the external ear, or pinna, although common in civilian life, are multiplied in time of war. Damaged ears produce such conspicuous deformities that every effort should be made to preserve all viable or questionably viable tissues. Later reconstructions, however painstaking the care with which they are wrought, never duplicate the normal ear. Prosthetic ears, although exact in contour and attractive for display purposes, have many drawbacks in daily use throughout a lifetime. Good early care can often prevent deformity, but when loss of substance has occurred, reconstructive surgery, by approximating normal, provides the most satisfactory correction of deformity. In exceptional cases, and where every vestige of the ear has been destroyed, prosthetic restoration may be indicated.

CRUSHING AND LACERATED WOUNDS

The ear is protected to some degree from physical injury by its resilient elastic-cartilaginous skeleton which recoils or bends away from a traumatizing

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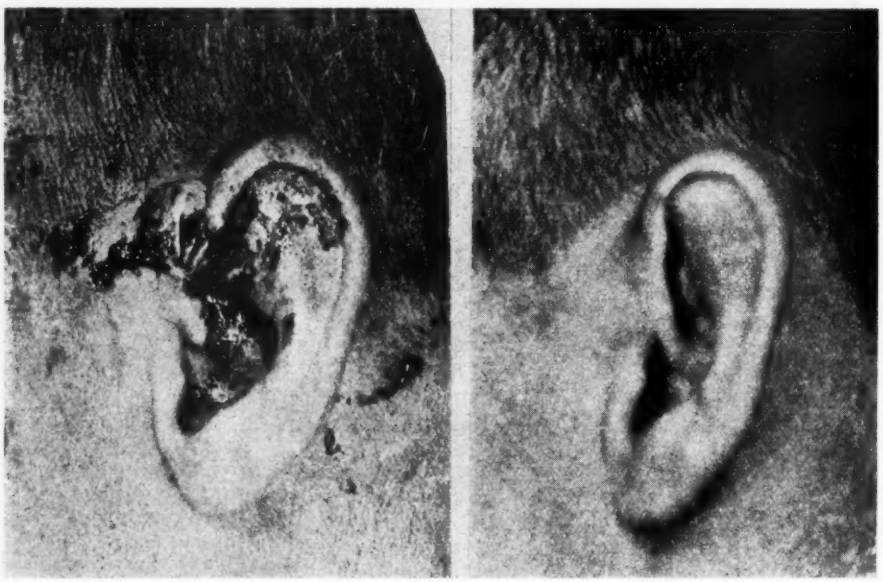
The opinions or assertions contained herein are the private ones of the writer, and are not to be construed as official or as reflecting the views of the Navy Department or the Naval Service at large.

force and thus diminishes the violence of the blow. Except for this meager protective mechanism, the ear is helplessly exposed to lacerating and crushing wounds of all descriptions. Blows, bites, cuts and stabs of civilian life have their battle counterparts in more destructive missiles, shrapnel, bayonet or sword wounds.

Simple crushing injuries result in the formation of a hematoma which should be evacuated aseptically through a tiny incision in the antihelix fold, followed by a pressure dressing. Failure of such hematomas to absorb or repeated injuries of this type give cauliflower ear, the late treatment of which is difficult.

Lacerations without loss of substance should be treated by cleansing, lavage, and conservative débridement. A "tongue-and-groove" joint of cartilage and overlying soft tissue is fashioned to prevent notching at the site of through-and-through lacerations (Fig. 1). This is done by trimming back the soft tissue on both sides of the ear cartilage of one fragment and resecting a corresponding bit of cartilage in the other fragment to form a "groove" into which the other cartilage will fit.



A. *B.*

Fig. 1.—*A*, Partial avulsion of left ear following truck accident; preoperative appearance. *B*, Postoperative appearance after conservative débridement and plastic repair by "tongue-and-groove" junction of wound edges.

When an ear is badly crushed, much of the covering soft tissue and cartilage may be destroyed. Here, one should avoid reckless destruction of good tissue and initial surgery should give questionably viable tags of skin a chance to recover. Continuous moist dressings of normal saline solution are applied and chemotherapy, with sulfadiazine and penicillin, is given as supportive treatment to minimize bacterial destruction of the injured tissues. Such an ear may heal with deformity, but structural preservation allows good late repair (Fig. 2).

One should be on the alert for damage to the middle ear and the initial injury or later complications should be treated promptly by appropriate measures. Associated skull fracture is common and leakage of cerebrospinal fluid from the ear should dictate a policy of noninterference, regardless of the condition of the pinna.

Fig. 2.—*A*, Appearance of crushed and lacerated ear on admission following injury by a cargo net. The bruised, blue tissues were covered with dirt, and cartilage was shredded. An associated cerebral concussion had occurred, but no skull fracture was present. *B*, Healed appearance following conservative débridement and normal saline solution dressings. *C*, All scar tissue excised and wound open. *D*, Skin loss replaced by full-thickness skin graft from behind other ear. *E*, Final result.

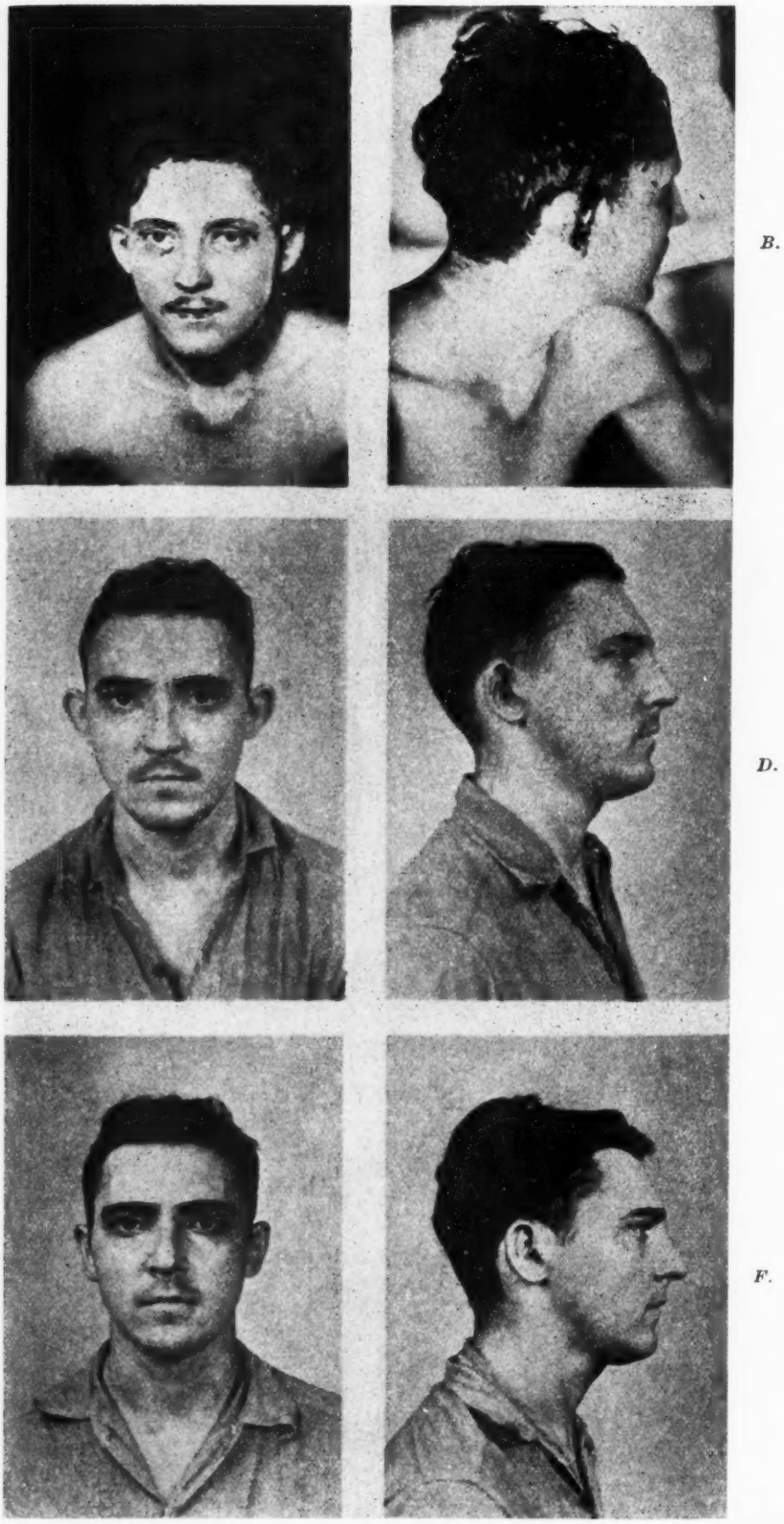


Fig. 3.—*A* and *B*, Appearance of ears, fourteen days following gasoline burn aboard motor launch. Initial pressure dressings have now been replaced by frequently changed normal saline dressings in preparation for grafting. *C* and *D*, Thick-split grafts have been applied and are now healed. Some contracture has occurred, however, and the ears have become slightly cup-shaped, protruding more than normal. *E* and *F*, Final appearance after plastic revision of the protruding ears.

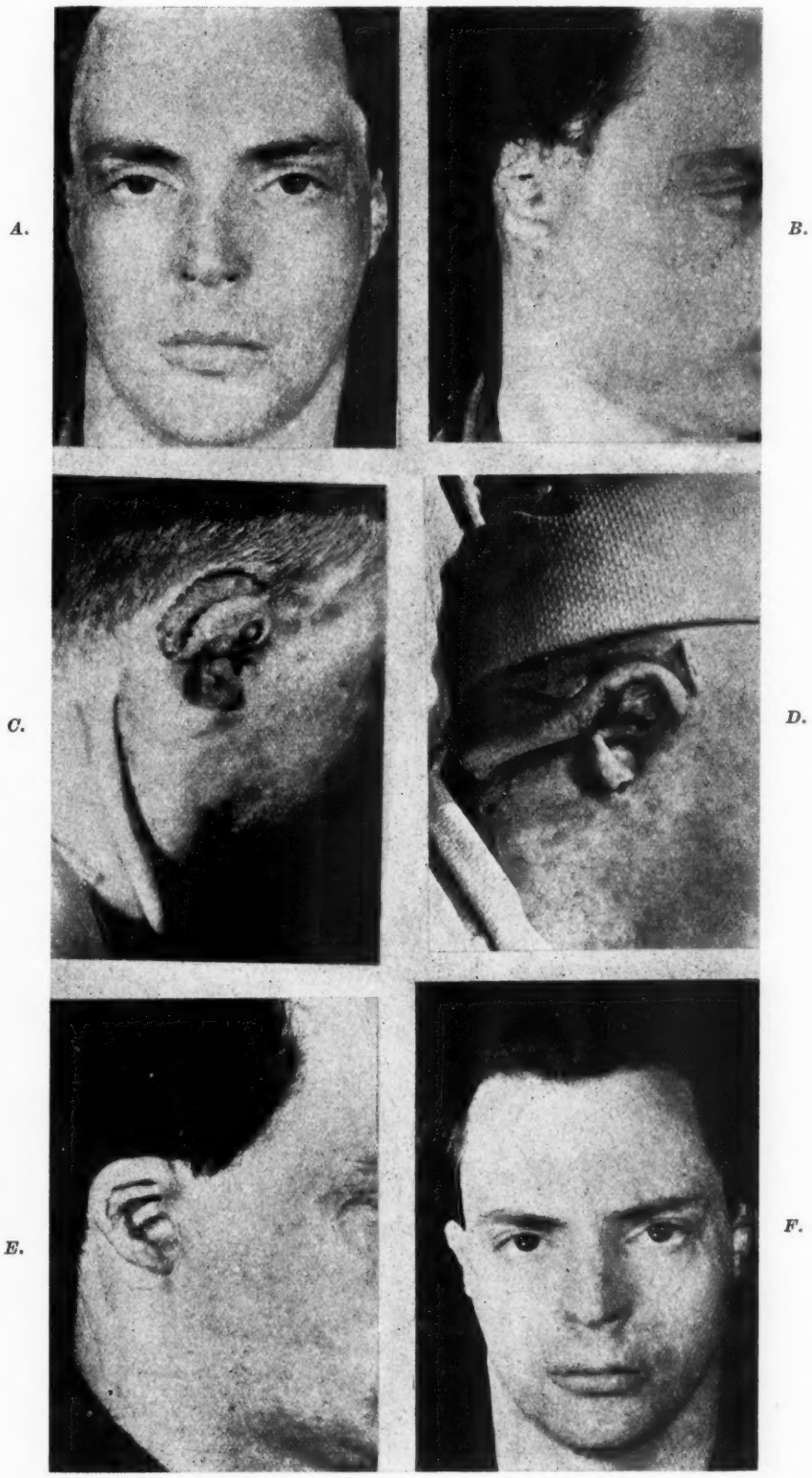


Fig. 4.—*A* and *B*, Subtotal loss of right ear following flash burn aboard torpedoed vessel. *C*, Retro-auricular flap has been swung forward and tubed pedicle flap formed on the neck. *D*, The tubed pedicle flap is now in place, reconstructing the helix; cartilaginous support is added later. *E* and *F*, Final result.

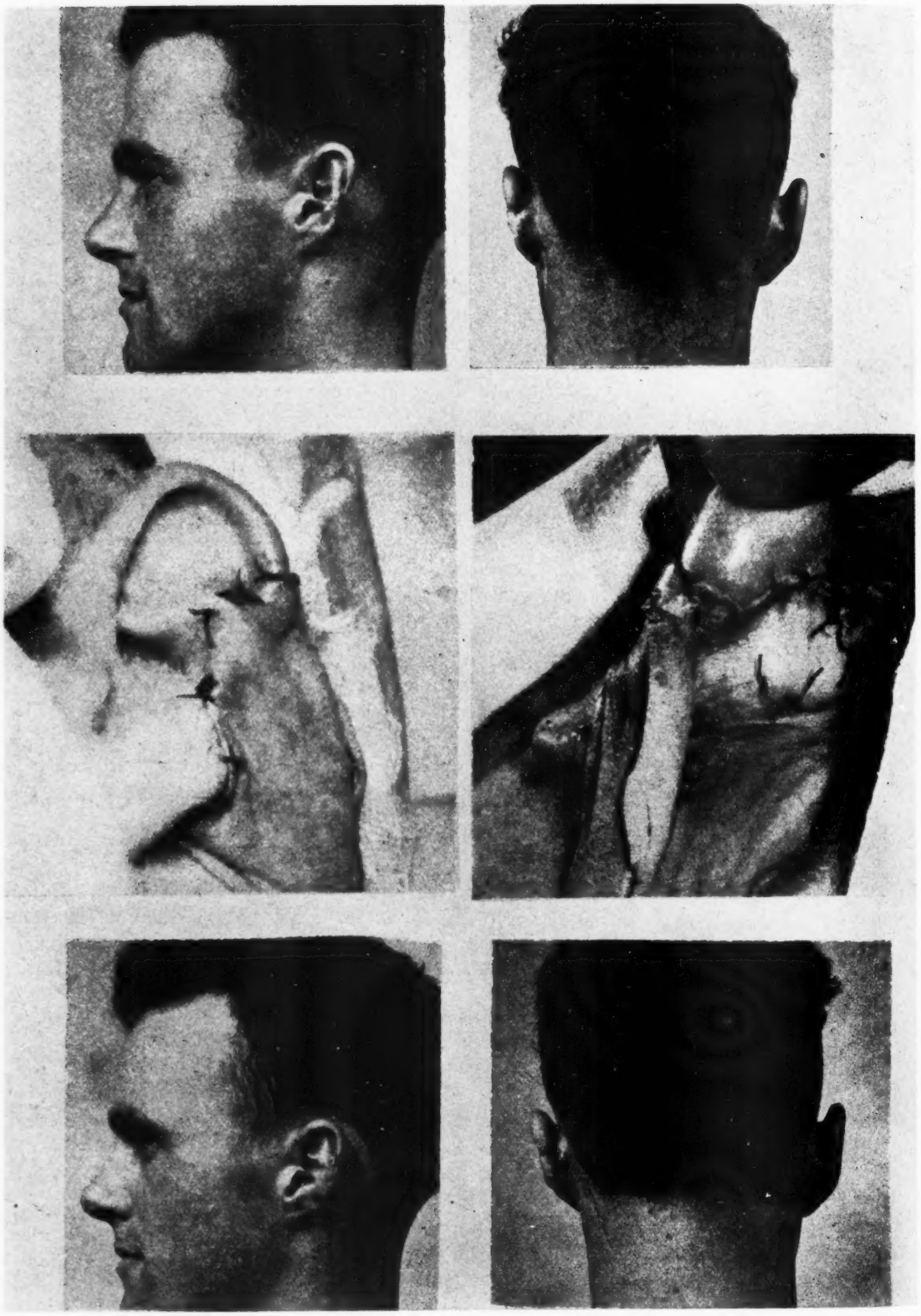


Fig. 5.—*A* and *B*, Necrosis following radiotherapy to hemangioma of left ear. Severely painful, ulcerating scar resulted. *C* and *D*, Retro-auricular flap has been applied following excision of all damaged tissue. A thick-split graft from the arm covers the defect behind the ear. *E* and *F*, Early end result at time patient returned to duty.

THERMAL OR CHEMICAL INJURY

Destruction of the external ear can occur from extremes of heat and cold, from electricity, and from the action of noxious chemicals. Although the latter two are less common, the familiar appearance of the ears of the burned aviator, tanker, or petroleum handler has become commonplace during this war. Extreme damage from frostbite is equally severe among exposed personnel.

Much of this thermal injury to the ear is so intensely destructive that no treatment can prevent deformity. If the injury is less severe, however, good care can conserve the surviving tissues. Nonadherent, fine-mesh gauze, impregnated with petrolatum or parawax, and mild pressure with carefully molded fluffed gauze should be used for the first few days. This prevents drying and further tissue necrosis. Thereafter, as soon as granulations appear the dressings are changed to fine-mesh gauze, wet with normal saline solution. These dressings are changed with sufficient frequency that all wound discharges are carried away and are not allowed to accumulate. The prompt application of free thick-split skin grafts will then prevent further contraction and give maximum preservation of tissues and normal appearance (Fig. 3).

In many cases extensive destruction of the pinna requires plastic surgical reconstruction. The usual methods of repair involve moving forward a flap of retro-auricular skin, with a subjacent thick-split graft, or the application of a cervical tubed pedicle flap of skin and subcutaneous tissue, or a combination of both of these procedures. Cartilaginous support, or metallic supporting wires or strips, are added at various stages in the procedure, according to the particular desires of the operator. The end results, although not normal, are usually quite acceptable (Fig. 4).

INFECTIONS AND TUMORS

Both pyogenic and granulomatous infectious processes can destroy the ear, but practically the only type with which we are concerned in this paper includes those infections which occur as a localized abscess or cellulitis following crushing wounds or burns. Systemic chemotherapy, with sulfadiazine and penicillin, is used to supplement local fomentations of warm saline solution. Early recognition and drainage of localized abscesses will prevent many of the contractures which follow infected wounds or burns of light severity. A number of these have been noted during World War II and their prompt recognition and treatment are urged.

Benign and malignant tumors are uncommon in the military age groups, but constant vigilance should be maintained for any suspicious lesions, particularly in the older, weather-beaten sailor. Their classification and treatment are beyond the scope of this paper, but one related traumatic agent, radiotherapy, may result in damage to the pinna that must be repaired (Fig. 5).

SUMMARY

In the surgical care of the injured external ear, all viable or potentially viable tissues should be conserved when the wound is débrided. Lacerations can be repaired primarily if there has been no appreciable loss of substance, or simple moist dressings may be applied to an injured ear which may require later reconstruction. Burned ears should be kept from drying and granulating surfaces grafted as soon as possible. Post-traumatic abscesses should be recognized and drained early.

HYPERTHYROIDISM WITH PROGRESSIVE EXOPHTHALMOS AND WITHOUT TACHYCARDIA

Report of a Case

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WE ARE reporting an unusual case of hyperthyroidism which has come under our observation, and which presented the following peculiarities: (1) absence of tachycardia; (2) severe progressive ophthalmopathy which dominated the clinical picture; and (3) absence of thyroid enlargement. The patient was successfully operated upon, and sections of the removed thyroid gland were reported as typical of Graves' disease.

CASE REPORT

C. S., a 45-year-old, dark-skinned Panamanian laborer, was admitted to the Amador Guerrero Hospital (under the care of M. R.), May 1, 1944, with the chief complaint of loss of weight, nervousness, and exophthalmos. He had considered himself in good health until a few months previously, when he began to notice a tired feeling after moderate effort, loss of weight in spite of good appetite, and protrusion of his eyes. He claimed to have lost thirty-five pounds in the preceding three months, and complained of nervousness, insomnia, excessive perspiration, and occasional diarrhea. But what bothered him most was the condition of his eyes, which were protruding, and subject to periodical bouts of edema, redness, and lacrimation with severe local pain.

Physical Examination.—Examination showed an undernourished, well-built, middle-aged man, in no acute distress. Temperature was 98.4° F., blood pressure 130/70, pulse rate 80. The heart was not enlarged, nor otherwise abnormal; the thyroid was not palpable. A slight tremor of the extended hands and fingers was noticeable. His eyes had a staring expression due to exophthalmos and widening of the palpebral fissure, blinking was almost abolished, and the patient was unable to close his eyes because of the exophthalmos, which made it impossible for the eyelids to cover all of the protruding eyeballs. At this time no edema or inflammation was present in the eyelids or conjunctivae.

During the first week in the Amador Guerrero Hospital, his weight was 102 pounds, basal metabolic rate plus 97, and later plus 92. Kahn blood test was negative; urine, blood counts, and x-ray of the chest were reported as normal. Electrocardiogram showed a normal sinus rhythm, with a rate of 80 beats per minute. The only electrocardiographic abnormality that was reported was a striking deformity of the P waves in Leads I and II, consisting in high, narrow, and peaked deflections.

During the first two weeks the patient was placed on a high-caloric, high-vitamin diet, with extra doses of vitamin B complex and vitamin C. He lost three additional pounds under this regimen. He began to complain of severe discomfort in the eyes, and developed redness, lacrimation, and moderate edema of the eyelids. An ophthalmologist was called in consultation to rule out intraocular primary pathology. The conclusion was reached that the whole picture could fit in the diagnosis of severe exophthalmos from hyperthyroidism. He was then put under iodine therapy, and a striking improvement took place in his general condition: nervousness disappeared almost entirely, bowel movements diminished from four to five to only one each day, he gained six pounds, and basal metabolic rate fell to plus 47. But, at the same time, the condition of his eyes became definitely worse. The edema of the eyelids became conspicuous, chemosis of the bulbar conjunctiva appeared, and pain and lacrimation interfered constantly with his sense of well-being. At this point the surgical department of the Amador Hospital was consulted on the advisability of immediate thyroidectomy, since we felt that medical treatment would not control the ophthalmopathy which was interfering

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with the patient's general condition, for constant pain and discomfort kept him restless and sleepless. No decision could be reached, and the patient continued under iodine medication, plus heavy sedation to control the pain in his eyes. The ophthalmopathy progressed unmercilessly until, June 20, a perforation of the left cornea developed, with loss of intraocular fluid. At this stage enucleation of the perforated eye was considered necessary, and the patient was transferred to the Santo Tomás Hospital.

Here evisceration of the left eye, and novocain block of the right stellate ganglion were performed, with temporary improvement in the condition of the remaining eye. His weight now was 106 pounds, basal metabolic weight plus 55, and electrocardiogram continued to show high and peaked P waves with normal sinus rhythm. Fasting blood sugar was 105, nonprotein nitrogen 29, blood cholesterol 126, blood calcium 11, blood Kahn negative, spinal fluid Kahn also negative. Urine was normal, and blood counts gave normal findings. Iodine medication was begun for the second time and he began to improve, gaining six pounds in weight, with a drop of the basal metabolic rate to plus 33. But here again doubts were expressed as to whether this patient really had hyperthyroidism, as most of the surgeons would not accept such a diagnosis with a pulse rate that never exceeded 90 per minute, and usually was between 60 and 80. Iodine therapy was accordingly stopped. In the next two weeks the patient lost six pounds, and the basal metabolic rate returned to plus 55, together with a definite exacerbation of the toxic symptoms. With this new evidence in favor of hyperthyroidism, iodine therapy was reinstituted and operation performed September 27, with the basal metabolic rate at plus 12, and weight at 117 pounds.

Fig. 1.

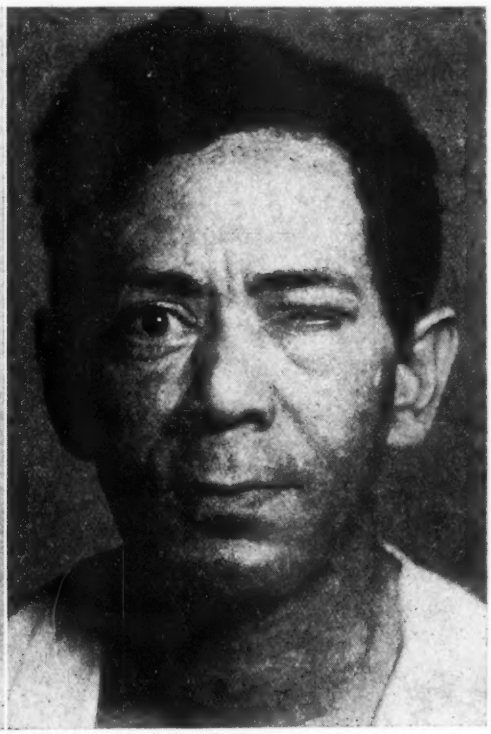

Fig. 2.

Fig. 1.—C. S., preoperative photograph taken in June, 1944, at Amador Guerrero Hospital.
Fig. 2.—C. S., photograph taken in December, 1944, three months postoperatively, at Santo Tomás Hospital, Panama.

He was transferred to the operating room under intravenous pentothal anesthesia, and operated upon under ethylene-ether-oxygen anesthesia. Subtotal right and total left thyroidectomy was successfully performed (G. O. C.). The removed gland was sent to the laboratory, and the following report was received: The specimen consisted of two pieces of thyroid, one weighing 19 Gm. and measuring 6 by 2 by 4 cm., the other weighing 20 Gm. and measuring 6 by 3½ by 2½ cm. The tissues showed congestion. The cut surface did not show formation of adenoma. The tissue appeared homogeneous and had a pink color slightly deeper than normal. On microscopic examination all sections showed the same pathologic picture; old and fresh hemorrhages were found, as were a few wandering cells in the interstitial tissues.

In general the acini appeared normal, but there were small areas that showed disappearance of colloid and changes in the shape of the epithelial cells. Careful examination did not reveal malignant changes nor small adenomas. Anatomic diagnosis was hypertrophy of the thyroid gland with changes which could be considered typical for toxic goiter.

The postoperative course was uneventful, except for a slight bronchitis that cleared in a few days under sulfadiazine medication. Immediately on returning to the ward he was placed under iodine and thyroid extract therapy (1 gr. thyroid, three times daily).

From then on his basal metabolic rate has been kept within normal limits, the condition of the right eye has improved gradually, and at present the conjunctival edema has disappeared completely, and he is even able to close the remaining eye, as the exophthalmos has disminished greatly. He has gained over thirty pounds in weight, and feels in perfect condition. No signs or symptoms of myxedema have developed. The basal metabolic rate in the last three months has been kept between plus 14 (Nov. 7, 1944) and minus 9 (Jan. 14, 1945). Weight on Jan. 13 was 138 pounds.

COMMENTS

The presence of progressive exophthalmos with periocular edema, chemosis of the conjunctiva, lacrimation, pain, and ulcerative keratitis, in the absence of tachycardia and of enlargement of the thyroid gland, made the case a difficult diagnostic problem for us. It might be assumed that nervousness, loss of weight, high basal metabolic rate, and gastrointestinal upsets could be due to his intense discomfort and mental strain brought about by the severe, progressive bilateral ophthalmopathy. The strongest evidence in favor of hyperthyroidism in this case was the observation that under iodine therapy all toxic symptoms decreased in intensity in spite of the little, or no, effect of this type of therapy upon the ophthalmopathy.

A similar case, in a 6-year-old Negro girl, has recently been reported by Haik,[1] with an extensive review of the literature on the subject of progressive exophthalmos in toxic disease of the thyroid gland. We feel that our case can be considered a typical example of Graves' disease of the ophthalmic type according to Means' classification.[2] In fact, our case presented the following characteristics:

1. Thyrotoxicosis was readily and repeatedly controlled by iodine medication.
2. The patient was a male.
3. Thyroid enlargement was not notable.
4. There was dissociation of thyrotoxicosis and ophthalmopathy, as general improvement of the toxic symptoms was often accompanied by an aggravation of the ophthalmopathy, and vice versa.
5. The more conspicuous ocular signs consisted of periocular edema and chemosis of the conjunctiva, although in our case (as in Haik's case) the classical ocular signs of exophthalmos, lid lag, and lid retraction were also present.

When thyroidectomy was decided upon, our main problem was to avoid the progression of the exophthalmos in the remaining eye, as we had observed that each time the basal metabolic rate had been lowered, through iodine medication, an increase in the severity of the ophthalmopathy resulted. Furthermore, most writers have warned that in such cases thyroidectomy usually brings about very little relief of the exophthalmos, and may even aggravate it.

Following the theory that considers exophthalmos a product of excessive secretion of thyrotropic hormone by the anterior lobe of the pituitary gland, and considers that this effect of the thyrotropic hormone is counteracted by the presence of thyroid hormone,[3] and knowing that the feeding of thyroid extract after thyroidectomy with the purpose of controlling the progression of the exophthalmos had already been tried,[4] we gave thyroid extract to the patient,

checking the basal metabolic rate at weekly intervals. In this case, this measure seems to have been successful, as in the three months that followed the operation, the right eye returned to almost normal condition.

Another interesting observation in this case was the peculiar high peaked P waves of the electrocardiogram. It has been common experience at the Santo Tomás Hospital to notice high P waves together with sinus tachycardia in cases of hyperthyroidism. This is the first instance in which the P waves were high and peaked with a normal sinus rhythm of 80 per minute. After the operation the P waves returned to a more normal configuration.

SUMMARY

We have presented a case of hyperthyroidism of the ophthalmic type. Enucleation of one eye (left) was necessary, because of perforation of the cornea following ulcerative keratitis. Thyroidectomy followed by thyroid administration seems to have given satisfactory results in the control of the ophthalmopathy.

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LOSS OF SCROTAL INTEGUMENT

A Simple Method of Repair

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THE primary purpose of this communication is to call attention to a simple method of stretching remnants of scrotal integument to an extent which may obviate the necessity of using more complicated plastic-flap operations.

Secondarily, a short résumé of a case history of scrotal gangrene following an operation for recurrent left inguinal hernia, in which the described method was used, will be presented.

A search of the literature on gangrene of the scrotum has failed to reveal a report of this condition following herniorrhaphy. There is no need here to discuss the general subject of scrotal gangrene, it being ably covered in numerous articles in general surgical and urological contributions.

The development of the gangrene in this instance followed a typical course, the cause, however, being evident rather than remote or unknown as is often the case.

CASE REPORT

F. K., a 44-year-old man, had an herniorrhaphy in May, 1937, for a direct, left inguinal hernia. There was recurrence one year later and a second operation was done. Three ten-inch strips were taken from the left fascia lata. All were used in making basket-weave repair. At the end of the first postoperative day, temperature of the patient was 101° F. Sulfanilamide was begun, 10 gr. (0.650) every four hours. There was a small amount

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of dark red drainage with white flecks from the wound. A culture was taken. Marked edema of scrotum and penis with ecchymotic discoloration was present. No edema was noted twelve hours before.

From this point on, a typical course of scrotal gangrene ensued, of the type described by Hinman.* The skin became reddened, tense, and glossy, and hot and tender. It then began to "sweat," becoming greasy and covered with an exudate, and a moist desquamation began. Within three days, sloughing scrotal integument was clipped away. The process extended until only a cuff of scrotum approximately one inch in length remained, a bit longer posteriorly. The testes and spermatic cords were bare but not involved in the infection. Two organisms were found in the culture; Beta hemolytic streptococcus and hemolytic *Staphylococcus aureus.*

At this time sloughs of fascial sutures were picked from the hernia wound. The herniotomy wound was irrigated with normal saline solution twice daily. The solution poured out over the exposed scrotal contents. The latter were also irrigated twice daily and then covered with petrolatum gauze. The testes were supported on a Bellevue bridge.

For eighteen days following the loss of scrotum, the upper wound drained in lessening amounts with strips of fascia lata coming away at various times. The wound closed on the twenty-first day. From then on the temperature remained normal, having ranged daily from 99 to 101° F. until this time. In the meantime, healthy granulation tissue had covered the testes and spermatic cords, the edge of the remaining scrotal skin becoming incorporated in this granulation tissue. The sulfanilamide had been discontinued after four days, since the patient felt that it nauseated him. (This was in 1938.)

Fig. 1.

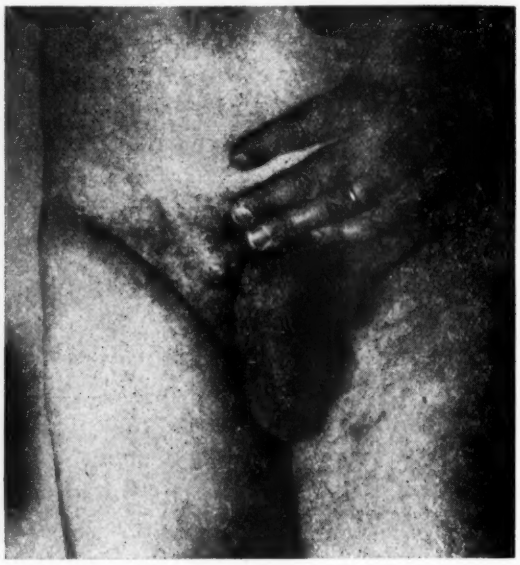

Fig. 2.

The personal equation in relation to the patient had much to do with the decision on the method of repair which was finally utilized. Surgeons will appreciate the mental agony which accompanies a complication such as developed in this case. The literature at that time, as today, proposed one of several methods of repair, all of which would no doubt have sufficed but which did not seem to fit the problem.

Undermining the contiguous areas of skin, including that of the thigh, seemed too dangerous from the standpoint of lighting up a latent infection. Waiting for epithelial outgrowth from scrotal remnant would have unduly prolonged an already stormy recovery, and there was little evidence that this would occur. Furthermore, the latter method of repair would leave a contracting cicatrix which, while covering the testes, would not be the best type of integument. Again, there was not enough scrotal skin to allow approximation of the edges, even had they been freed and undermined. Certainly, if possible, the patient was to be saved the nuisance of an operation requiring the use of plastic flaps from the insides of the thighs, which had been thought of and suggested as the safest procedure.

*Hinman, Frank: The Principles and Practice of Urology, Philadelphia, 1935, W. B. Saunders Company.

At this point, recalling the enormous extent to which scrotal skin with its dartos muscle becomes attenuated in accommodating large hernias, the thought occurred that some method of stretching the skin might serve the purpose. This was accomplished in the following simple manner.

Two strips of adhesive plaster were cut to the width of the scrotal skin remaining on either side, at the root of the penis. Their length was such that the strips could be brought down and, with some tension, fixed to the inner sides of the thighs, each strip being brought to the side opposite its upper end. Thus it was found possible to keep enough tension on the scrotal remnant to stretch it gradually yet not disturb the patient.

The edges of the scrotal skin remained in their fixed position but the parts above gradually came down over these edges. The adhesive was changed when it softened in its upper portion, and the stretching was increased by placing straps lower on the thighs each time. This was done daily, sometimes every second day. Looking back, it was probably not necessary to continue this stretching for three weeks, but the progress was so remarkable that it became apparent that an almost normal scrotum was in the process of formation. When the skin had stretched to the point at which it was considered sufficient, the edges were freshened and the well-stretched integument sutured over the exposed contents which at this time were completely covered by healthy granulation tissue.

It was forty days after the first slough came away that the skin edges of the now-stretched scrotum were freed from the granulation tissue. The latter was left intact. Oozing was controlled by hot saline solution packs. The freshened skin edges were then approximated by a running suture of alloy steel wire, the ends of which were twisted together over a gauze dressing. Healing was per primam, the patient leaving the hospital seven days later; the wire was removed at the end of two weeks.

Five years later, the scrotum is small but perfectly normal in appearance and action. There has, strangely enough, been no recurrence of the hernia.

SUMMARY

1. A simple method of repair of the scrotum is presented. It may be tried whenever there is enough remnant of scrotal integument to which adhesive plaster (or other means which would produce tension) may be applied.

2. A case of fulminating scrotal gangrene following a fascia-lata-strip repair of recurrent/inguinal hernia is cited.

3. The method might well be tried in war wounds of the scrotum. It seems that patients might be spared the application of more extensive plastic procedures.

My associate Lieutenant Colonel Floyd R. Parker, Medical Corps, Army of the United States, carried out the meticulous aftercare in the case and deserves a good share of the credit for the final outcome.

Book Reviews

Care of Neurosurgical Patient. By Ernest Sachs, M.D., St. Louis, 1945. The C. V. Mosby Company.

The author has written, very interestingly, a most instructive text. The treatment of the neurosurgical patient is concisely but thoroughly considered. Beginning with the preliminary examination, the treatment of the patient is followed through the preoperative care, the operative technique, and the postoperative care. On pertinent controversial points such as diagnostic measures and surgical approaches, Sachs reviews the methods in question. He then points out very adequately why his recommendations are the more sound.

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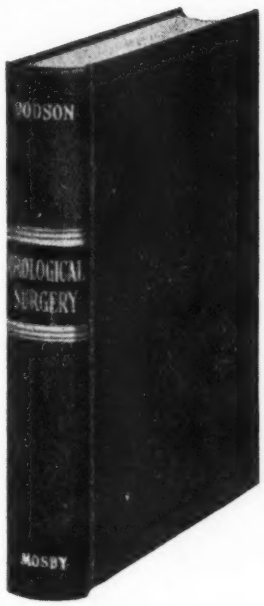

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